Some effects of hippocampal lesions on the behaviour of pigeons

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SOME EFFECTS OF HIPPOCAMPAL LESIONS ON THE BEHAVIOUR OF PIGEONS

by

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ABSTRACT

The behavioural effects of hippocampal lesions in birds have not previously been investigated, although there is now considerable evidence, which is reviewed here, of structural and histochemical similarities between the avian and the mammalian hippocampus. Therefore, a series of experiments were carried out to study the effects of hippocampal lesions in pigeons, and it was found that they performed more efficiently on both the acquisition and reversal of a 70:30 colour probability discrimination, confirming a prediction derived from the cognitive mapping theory of hippocampal function (O'Keefe and Nadel, 1978). Hippocampal pigeons were also impaired on the serial reversal of a spatial discrimination and on a DRL 10 schedule of reinforcement, but not on the acquisition or the reversal of a simultaneous visual form discrimination, a delayed spatial alternation task, or a delayed colour alternation task. Furthermore, they did not show increased resistance to extinction, except following DRL 10 training, or increased response perseveration in reversal. These effects show many similarities to those that have been found to occur in hippocampal mammals in comparable tasks, and it is proposed, therefore, that the results of the experiments reported in this thesis provide good evidence that the avian hippocampus and the mammalian hippocampus are behaviourally homologous. These results extend the findings by others of structural similarities between the hippocampus in birds and mammals and therefore lend considerable support to the proposal that they are homologous structures. Moreover, in common with much of the mammalian hippocampal data, the present results do not support the response-inhibition, response-shift, or selective attention theories of hippocampal function, but it is argued that they support instead the hypothesis that the hippocampus is involved in the processing of spatial information, and that they are consistent with the cognitive mapping model of the hippocampus proposed by O'Keefe and Nadel (1978).

INTRODUCTION

Various arguments have been presented in the past for the value of comparative studies, and recently some of these have been reaffirmed. Hodos (1974) has pointed out that the comparative approach allows the study of the diversity and generality of phenomena in nature, can provide clues to trends in evolution, and can play a particularly important role in the establishment of relationships between structure and function. Also, Macphail (1975b) has argued that our understanding of the mammalian brain may well be aided by the study of a species in which the organisation of the brain differs from that of mammals and which is capable of a high degree of learning. Two such candidates, he proposes, are birds and fish.

Like the rat, the pigeon has been found to be a particularly useful animal in behavioural studies. Pigeons are reasonably small, are relatively inexpensive to obtain (although perhaps they are not quite as readily available here as they are in the U.S.A., or as rats are), and are easy to house and maintain. They are also easy to handle, to motivate, and to train, and they have excellent visual acuity and colour vision. Thus, they are very suitable animals for the study of various motivational, learning, memory, and perceptual processes, and it is for these reasons that pigeons have been widely used by psychologists in laboratory studies of animal behaviour. However, compared with the mammalian brain, surprisingly little is known about the behavioural functions of the avian brain, and it has been suggested that this is simply because man is a mammal, and therefore it has been argued that insight into the functioning of the human brain is much more likely to be gained from studying the brain of the rat rather than the brain of the pigeon.

During the past 10 to 15 years there appears to have been a change of attitude towards work with birds, and there have been a number of anatomical, electrophysiological, and behavioural studies of the avian brain which have provided a

valuable basis for further studies of brain function in birds. Besides demonstrating some remarkable correspondences between regions and pathways in the avian and mammalian brains, they have helped to establish the hyperstriatal complex in birds as an area of considerable interest. The hyperstriatal region is a complex structure with apparently diverse functions, there being evidence to suggest that, separately, parts of the hyperstriatal complex may be comparable with visual cortex, limbic cortex, and part of the pyramidal system in the mammalian brain. Altogether there have been relatively few studies of the behavioural effects of hyperstriatal lesions, and apart from experiments by Macphail (1971, 1975a, 1976a, 1976b) and Hodos, Karten, and Bonbright (1973), who made their lesions electrolytically, the majority of the studies have used vacuum aspiration techniques, and therefore the lesions that were made were usually moderately large and fairly imprecise. It is, perhaps, of interest to note that, as far as lesion studies are concerned, current approaches to the study of this region of the avian brain are still at the stage that lesion studies of frontal and temporal lobe function in monkeys were at in the late 1930's (e.g., see Iversen, 1973). At that time large amounts of tissue were removed from either of these two areas and the effects on behaviour studied (Jacobsen, 1935, 1936; Klüver and Bucy, 1937, 1939), and then gradually, investigators began to make smaller and more localised lesions in order to study the functions of the smaller structures that had been included in the earlier, more extensive, lesions. Because of the heterogeneous nature of the hyperstriatal complex, there is now a need for its different regions to be studied by means of small, precise, electrolytic or radiofrequency current lesions in order to provide more detailed behavioural evidence which, hopefully, will support the anatomical and electrophysiological evidence that is currently available.

Since there have not been very many studies of the behavioural effects of hyper-

striatal lesions, only a relatively small range of behavioural tasks have so far been employed. Nevertheless, hyperstriatal lesions have been found to produce some behavioural changes that have been likened to some of the effects that occur in mammals following hippocampal lesions (Macphail, 1969-1975b; Stettner, 1974). However, the avian hippocampal formation lies adjacent to part of the hyperstriatal complex, and in several studies has been included inadvertently in lesions of the hyperstriatum. This therefore suggests the possibility that the hippocampal damage may have contributed to the hippocampal-like effects that have been observed. Although there have been several studies in which the lesions were restricted to the hyperstriatal region, leaving the hippocampus undamaged, hippocampal-like deficits have been found on only two types of task (see chapter 1, pp. 22-29). However, performance on these tasks has also been shown to be affected by lesions in other regions of the mammalian brain, apart from the hippocampus, suggesting, therefore, that comparisons between the behavioural effects of lesions of the avian hyperstriatal complex and the mammalian hippocampus ought, perhaps, to be viewed with some caution.

Although a certain amount of interest in the mammalian hippocampus was created by the work in the late 1930's of Papez (1937) and Klüver and Bucy (1939), it was not until the 1950's that this interest really flourished, as a result of the reports by Scoville (1954), Terzian and Dalle Ore (1955), Scoville and Milner (1957), and Penfield and Milner (1958) of profound memory disturbances in patients who had undergone the surgical removal, bilaterally, of part of the temporal lobe, including the uncus, amygdala, and most of the hippocampus. However, early attempts to reproduce the memory disturbance in mammals with bilateral hippocampal lesions were remarkably unsuccessful. Various experiments (e.g., Isaacson, Douglas, and Moore, 1961; Kimble, 1963; Kimble and Pribram, 1963; Wickelgren and Isaacson,

1963; Teitelbaum, 1964; and Webster and Voneida, 1964) showed that rats, cats, and monkeys with hippocampal lesions were able to learn a variety of tasks at normal rates. Nevertheless, a number of behavioural changes have been found to occur on certain tasks, and these include impaired passive avoidance learning (Kimura, 1958; Isaacson and Wickelgren, 1962), improved active avoidance learning (Isaacson et al, 1961; Green, Beatty, and Schwartzbaum, 1967), increased resistance to extinction (Niki, 1965; Peretz, 1965), impaired reversal learning (Thompson and Langer, 1963; Silveira and Kimble, 1968), impaired successive go, no-go discrimination learning (Buerger, 1970; Woodruff, Means, and Isaacson, 1973), reduced distractibility to novel stimuli (Wickelgren and Isaacson, 1963; Hendrickson, Kimble, and Kimble, 1969), reduced or absent exploratory behaviour (Leaton, 1967; Nadel, 1968), impaired spontaneous alternation (Roberts, Dember, and Brodwick, 1962; Stevens, 1973b), impaired learning of complex mazes (Thomas and Otis, 1958; Jackson and Strong, 1969), and reduced ability to respond at normal rates on certain schedules of reinforcement (Clark and Isaacson, 1965; Jarrard, 1965). Other reports, many more recent than most of these, have confirmed all of these findings, although there are also reports in which deficits were not found on many of these tasks (for a recent, very comprehensive review, see O'Keefe and Nadel, 1978).

Such a variety of deficits, not surprisingly, has given rise to a variety of explanations that have been proposed in an attempt to account for these effects. Indeed, Elmes, Jarrard, and Swart (1975) have suggested that "the behavioural changes following damage to the hippocampus are only slightly more numerous than the theories postulated to account for hippocampal function" (p. 51). However, a pattern of behaviour that did emerge as a common characteristic of the effects of hippocampal lesions was the repetitiveness of responses, and this has become known as perseverative behaviour, or response perseveration. Consequently, one hypothesis

was that the hippocampus is involved in response inhibition (Kimble and Kimble, 1965; McCleary, 1966), an idea which recently has been presented again, in a slightly modified form (Altman, Brunner, and Bayer, 1973). Alternative versions of the inhibition concept have proposed that the hippocampus plays an important role in the generation of Pavlovian internal inhibition (Kimble, 1968; Douglas, 1972), or in the inhibition of attention (Douglas and Pribram, 1966; Douglas, 1967; Kimble, 1968; Silveira and Kimble, 1968; Kimble and Kimble, 1970). In addition, these last two studies also discussed the effects of hippocampal lesions in terms of impaired hypothesis behaviour, and subsequently Isaacson and Kimble (1972) proposed that the hippocampus is involved in the regulation of hypotheses, a view also expressed earlier by Pribram, Douglas, and Pribram (1969), and more recently by Stevens (1973a). Related to this is Olton's (1972a) proposal that the hippocampus is part of a response-shift mechanism.

It was noted above that there have also been a number of reports in which various of these deficits were not found following hippocampal lesions in mammals. In a number of studies it has been shown that hippocampal mammals are capable of normal levels of response inhibition (Winocur and Salzen, 1968; Olton, 1972a; Samuels, 1972; Stevens, 1973c; Elmes et al, 1975; Nadel, O'Keefe, and Black, 1975; Winocur and Black, 1978; Plunkett and Faulds, 1979), and also do not show impaired attention (Schram, 1971; Harley, 1972; Olton, 1972a; Samuels, 1972). These findings therefore raise serious problems for the inhibition hypotheses of hippocampal function. Considerable concern has also been expressed over the discrepancies that appeared to exist between the human and the animal data, which, as O'Keefe and Nadel (1978) point out, suggested to a number of investigators the possibility that there were major differences in hippocampal function between humans and animals. Nevertheless, Weiskrantz and Warrington (1975) argued that these discrepancies must

be the result of "only one or a combination of three possibilities: either the description of the defect in man is incomplete or inadequate, or the appropriate methods of analysis have not yet been discovered for the animals, or man and other primates are fundamentally different in the expression of brain function even though neuroanatomically the relevant regions of the brain are so very similar" (p. 411). A reappraisal of both the human and the animal experimental data (Weiskrantz, 1971; Weiskrantz and Warrington, 1975) has shown that the last of these three possibilities is rather less probable, and Weiskrantz and Warrington (1975) have proposed that the hippocampus, in both animals and man, plays a major role in reducing interference effects, thereby enabling the retrieval of appropriate information and/or the selection of appropriate responses. Evidence that supported this proposal came from various experiments (see Weiskrantz and Warrington, 1975) in which the use of partial cueing techniques in amnesic patients was found to be particularly effective in enabling the successful recall of material that otherwise was believed to have been forgotton. Also consistent with this hypothesis are the results of a recent experiment by Winocur (1979), in which he found that the acquisition and retention of a visual pattern discrimination were more impaired in hippocampal rats by high interference tasks than they were in normal and cortical control rats.

A further recent animal experiment that supports this proposal is that of Winocur and Black (1978), in which they showed that hippocampal rats, trained 24 hours earlier on a passive avoidance task in a runway, could show normal recall of the task provided they were given appropriate partial cueing. However, it also supports an alternative explanation, which is provided by the spatial information processing, or cognitive mapping, model of O'Keefe and Nadel (1978). In this it is proposed that the normal animal explores its surroundings, and from the information it gains, and with the aid of the hippocampal cognitive mapping system, it is able to generate a

cognitive map of its environment. This then allows the animal to use place hypotheses, i.e., to use spatial cues or information, in its learning of a variety of tasks. This they refer to as the locale system. In addition there are the taxon systems, which involve most of the rest of the brain (i.e., excluding the hippocampus), and which allow the animal to use guidance and orientation hypotheses. These can be thought of as S-R-S chains, in which guidance hypotheses are concerned with the value of cues or events and orientation hypotheses relate to the responses that are required. It is further proposed that, whereas place hypotheses allow for flexible and rapid changes in behaviour and the retrieval of context-dependent information, as a result of which they are not especially susceptible to interference effects, guidance and orientation hypotheses result in rigid and persistent behaviour patterns, do not allow the use of information relating to spatial location, and are particularly prone to confusion, or interference between behaviours that are appropriate to different contexts.

Observations of discrimination learning in animals suggests that they first learn where to respond, and only later to what. Thus Means and Douglas (1970) showed that, interpreted in terms of the cognitive mapping model, normal rats trained on a spatial task in a +maze initially used place hypotheses, and with continued training switched to using other types of hypothesis. However, since animals with hippocampal lesions have been deprived of their cognitive mapping system, they are unable to gain information about their environment through exploration, and consequently are unable to form cognitive maps and thus to make use of place hypotheses. As a result they are totally dependent upon their taxon systems and, in the case of initial learning about a problem, the disadvantages that go with them. It should be expected, therefore, that hippocampal animals would largely be impaired on tasks in which the use of place hypotheses are important, and should perform as well as normal animals in situations which rely entirely on the use of guidance and orientation hypotheses.

Besides the observation by a number of investigators (e.g., Mahut, 1971; Samuels, 1972) that the spatial aspects of a task appear to be especially important in the deficits shown by hippocampal animals, a number of experiments have been carried out recently, explicitly to investigate place learning in hippocampal rats (e.g., Plunkett, Faulds, and Albino, 1973; O'Keefe, Nadel, Keightley, and Kill, 1975; Olton, Walker, and Gage, 1978), and the results of all of these experiments support the hypothesis that the hippocampus is involved in the processing of spatial information. The results of another experiment, carried out recently by Sinnamon, Freniere and Kootz (1978), also support this hypothesis, but additionally show similarities to some aspects of the amnesic syndrome in humans.

Both Weiskrantz and Warrington (1975) and Nadel and O'Keefe (1974) have argued that it now does not seem that there are major functional differences between the hippocampus in man and in other mammals. Nauta and Karten (1970) also point out that "the limbic system has had a fairly stable evolutionary history" (p. 10), and Angevine (1975) proposes that, on the basis of what we now know about its comparative anatomy, the hippocampal region would seem to be consistent with this statement. Nevertheless, he recommends caution in the light of our ignorance of this region in many vertebrates, and in his brief discussion of the avian hippocampus, he suggests that a further problem in birds is their divergent evolution from the extinct stem reptiles compared with the mammals. However, there is now a reasonable body of evidence that shows that there are considerable structural and histochemical similarities between the avian hippocampus and the reptilian hippocampus on the one hand and the mammalian hippocampus on the other (see Chapter 1). It is proposed here, then, that the finding of functional similarities between the avian and the mammalian hippocampus, as shown by similar effects following lesions in this structure, would lend further considerable support to the notion that they are homologous structures.

The plan of this thesis is therefore as follows: Chapter 1 presents a review of the various studies of the avian brain that are relevant to the study of the effects of hippocampal lesions in birds, there being no previous work precisely on the behavioural effects of lesions of the avian hippocampus. Details of the experimental method, and the surgical and histological procedures that were used in this study are presented in Chapter 2. The various experiments that were carried out are described in Chapters 3 to 9; and finally, Chapter 10 presents a general discussion of the results of these experiments and some conclusions.

CHAPTER 1 Comparative Aspects of the Avian Hippocampus

Introduction

Very little is known about the behavioural functions of the avian hippocampus. To the writer's knowledge, no exactly comparable work, involving small lesions restricted primarily to the hippocampal and parahippocampal areas only, has been reported previously, although three somewhat related studies have been published (Benowitz, 1972; Benowitz and Lee-Teng, 1973; Lee-Teng and Sherman, 1969), and several studies have been reported in which birds with hyperstriatal lesions also received damage to the hippocampal formation (Reynolds and Limpo, 1965; Stettner and Schultz, 1967; Macphail, 1969).

In contrast, there now exists a reasonable body of information concerning the morphology and anatomy of the avian hippocampus which, despite the differences in the organisation between avian and mammalian brains, bears comparison with the mammalian hippocampus. On the basis of this there have been various proposals that the avian and the mammalian hippocampal formations are homologous. However, it has been argued (Zeigler, 1963a; Campbell and Hodos, 1970) that the demonstration of structural similarities are of questionable value until they can be supported by evidence of functional similarities.

Despite the lack of direct behavioural evidence on the avian hippocampus, it is argued here that, by comparing the behavioural effects of combined hyperstriatal and hippocampal lesions with those produced by lesions restricted to the hyperstriatal complex, certain inferences may be made concerning the possible effects of hippocampal lesions in birds, and it is found that these effects are similar to those produced by hippocampal lesions in mammals. Nevertheless, while providing a useful guideline, they can only be regarded as hypotheses in need of testing, and evidence to support these hypotheses can only be obtained from behavioural studies in which lesions are

restricted to the avian hippocampal formation. Similarities between these findings and those obtained from behavioural studies of the mammalian hippocampus would then provide considerable support for the proposed homologies between the avian and the mammalian hippocampal formation that, at present, are based on structural grounds alone.

In this chapter, following a brief description of the avian forebrain, behavioural, anatomical, and electrophysiological studies of the avian hyperstriatal complex and related structures are discussed in detail. This is followed by a selective review of the behavioural effects of hippocampal lesions in mammals on tasks similar to those that have been presented to hyperstriatal lesioned pigeons. Finally, the comparative anatomy of the hippocampal formation in mammals, birds, and reptiles is presented, followed by a consideration of the evolutionary relationships between these three orders, and the question of homology.

The anatomy of the avian forebrain

That the organisation of the avian forebrain differs markedly from that of the mammalian brain has been known for many years. Externally, perhaps the most noticeably different feature of the avian brain is the large, laterally displaced optic lobes (see Figure 1). The major difference, however, lies in the internal structure of the forebrain. In the mammalian brain the cerebral hemispheres are composed of numerous neural structures and cell groups, together with considerable numbers of myelinated fibre tracts, a large proportion of which interconnect corresponding regions of the two hemispheres via the corpus callosum, surrounded by a large expanse of multilayered tissue, the neocortex, a development which is unique to mammals. In contrast, the avian cerebral hemispheres consist largely of what traditionally, but misleadingly, has been referred to as the corpus striatum, or the striatal complex, which is composed of five large nuclear masses situated between the medially placed ventricle and the lateral

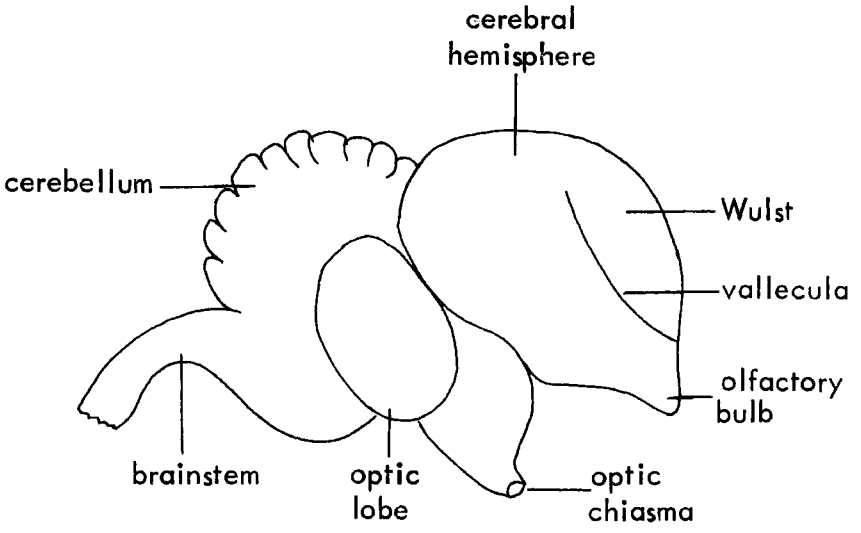


Figure 1. Lateral view of the brain of the pigeon.

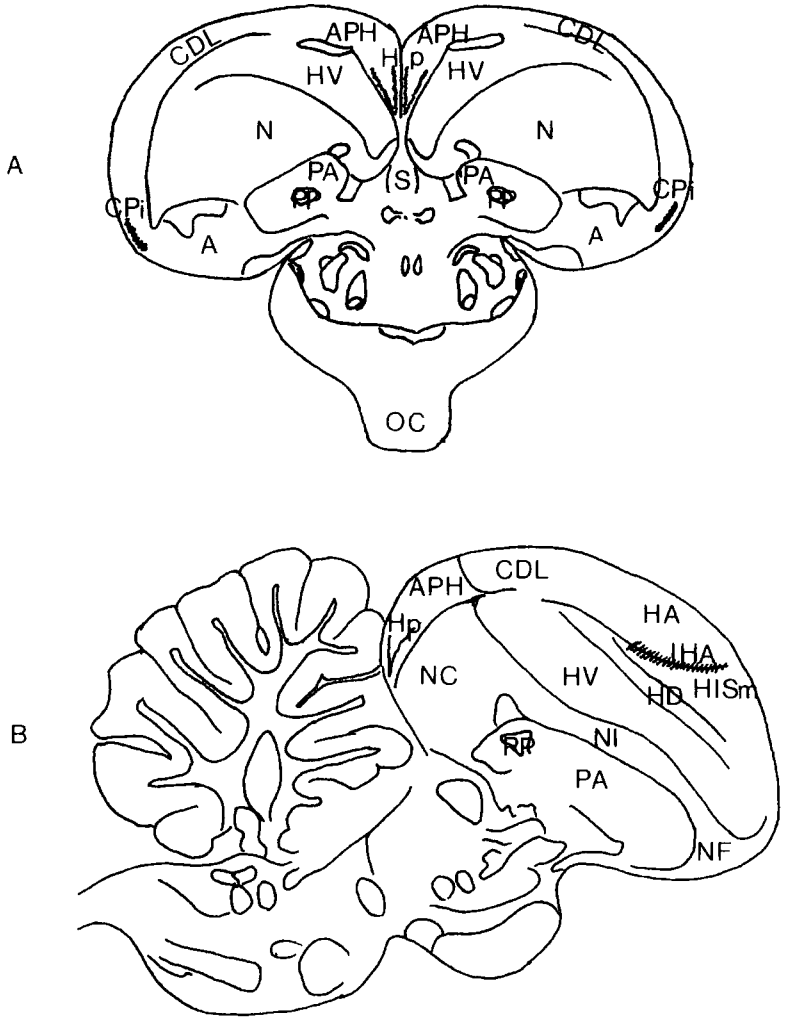


Figure 2. A. Coronal section (A7.25), B. Sagittal section (L2.00) of the pigeon brain (redrawn from Karten and Hodos, 1967). For abbreviations see text.

wall of the hemisphere. These five regions, which are differentiated mostly on the basis of their cytoarchitecture, are the paleostriatum, archistriatum, neostriatum, ectostriatum, and hyperstriatum (see Figure 2).

The paleostriatum, which is in the ventral part of the hemisphere, is divided into the paleostriatum primitivum (PP) and the paleostriatum augmentatum (PA), and in a ventrolateral position, forming the wall of the posterior third of the hemisphere, is the archistriatum (A). Overlying the paleostriatum, and separated from it by the lamina medullaris dorsalis, is the neostriatum (N), the largest of the striatal bodies. It extends from the posterior pole of the hemisphere and gradually decreases in size towards the anterior pole, and is usually divided into three distinct regions, the frontal neostriatum (NF), the intermediate neostriatum (NI), and the caudal neostriatum (NC). Also lying above the lamina medullaris dorsalis, and largely surrounded by the neostriatum, is the ectostriatum (E). Lying dorsal to the neostriatum and separated from it by the lamina hyperstriaticus is the hyperstriatum, or hyperstriatal complex, a structure which is unique to the avian brain. It is divided into the ventral hyperstriatum (HV), the dorsal hyperstriatum (HD), the hyperstriatum intercalatus suprema (HISm), the intercalated nucleus of the accessory hyperstriatum (IHA), and the accessory hyperstriatum (HA). Together these latter four structures form the sagittal elevation, or Wulst, whose lateral extent is bounded by the vallecula (compare Figures 1 and 2B).

Between the ventricle and the medial surface of the hemisphere, in the ventral part of the medial wall, lies the septal area (S). In the dorsomedial and dorsal regions of the medial wall is the hippocampal formation (Hp), and dorsolateral to this is the parahippocampal area (APH), which becomes continuous, caudally, with the corticoid tissue of the dorsolateral surface of the hemisphere (CDL), and rostrally, with HA, i.e., part of the Wulst.

Up until the late 1930's there was still some controversy concerning the location

and extent of cortical tissue in the avian brain. It is probable that this was due to a combination of factors, most prominent of which, no doubt, was the considerable variation in cortical development amongst different species of birds, and the general lack of agreement over the definition of cortex. In 1909, Brodmann proposed that cortex be sub-divided into two types: homogenetic, which is six-layered and is otherwise known as neopallium or neocortex, and heterogenetic, which does not have six layers and is therefore phylogenetically older. For this reason it is also known as archipallium or archicortex. On the basis of this definition Rose (1914) argued that, since six-layered cortex is clearly not present in the avian brain, birds do not have a neopallium, but that archipallium is represented in the areas he designated the hippocampus and the entorhinal area. Even so, Huber and Crosby (1929) and Ariëns Kappers, Huber, and Crosby (1936), despite subsequent work, were not convinced that true laminated, and therefore cortical, tissue was present in the avian brain. And in 1939, according to Pearson (1972), although the embryological work of Kuhlenbeck (1938) supported the earlier proposals of the existence of archipallium, Crosby remained sceptical (Crosby and Humphrey, 1939). It was only after Craigie's later anatomical studies (1934-1940: see Pearson, 1972) that the question of avian cortex was resolved. It is now accepted that cortical tissue occurs in the medial, dorsal, and lateral areas of the brain, and that it is phylogenetically older cortex, i.e., allocortex. It comprises, respectively, the hippocampal and parahippocampal areas, the dorsolateral corticoid area, and the periamygdalar and prepyriform areas.

For many years, therefore, there have remained the questions of the avian homologue of the mammalian neocortex, and of the functions of the avian cortical areas. In 1958, despite the earlier findings, particularly those of Craigie and Kuhlenbeck referred to above, Stingelin proposed that the Wulst was homologous with the mammalian neocortex. Then, in 1960, Cobb reported that, in very general terms, there appeared to be a

positive relationship between the overall size of the hemisphere, relative to the size of the brainstem, and 'intelligence' or adaptability in a number of species of birds. Furthermore, he found that it was mainly the hyperstriatum, and particularly the Wulst, that varied in the different types of birds, and that it appeared to be large in the more 'intelligent' species. Since the Wulst and the mammalian neocortex are not, in fact, homologous, Cobb concluded that "the Wulst of birds and the neocortex of mammals may have similar functions and thus they may be analogous organs" (Cobb, 1960, p.407). Over the past ten to fifteen years a number of workers, and notably Karten and Hodos and their colleagues, have obtained anatomical, electrophysiological, and, to a lesser extent behavioural, evidence which provides good support for this proposal, at least as far as certain sensory functions are concerned (for recent reviews, see Cohen and Karten, 1974, and Salzen and Parker, 1975). However, the role of the avian cortical regions, and in particular the hippocampal and parahippocampal areas, is a topic which has been almost completely overlooked.

Experimental studies of the avian forebrain

It was stated earlier that no reports have yet been published in which are described the effects of lesions restricted to the hippocampal complex in birds. However, as Salzen and Parker (1975, pp. 215 and 235) have themselves pointed out, lesions of the hyperstriatal complex have commonly involved varying amounts of damage to the parahippocampal and hippocampal areas. Also, three studies have been reported in which the hippocampal and parahippocampal areas were specifically included in lesions of the dorsomedial hyperstriatal region in chicks (Benowitz, 1972; Benowitz and Lee-Teng, 1973; Lee-Teng and Sherman, 1969). Thus, it could be argued that at least some evidence is available concerning the effects of hippocampal lesions in birds, although it is necessarily confounded to a greater or lesser extent by the effects of the hyperstriatal damage. There are, however, several reports of studies of the behavioural effects of

lesions restricted to the hyperstriatal complex, leaving the hippocampal and parahippocampal areas intact, and a number of anatomical and electrophysiological studies of the hyperstriatum, and these various studies are therefore reviewed here.

Behavioural effects of hyperstriatal lesions

a) Lesions that include the hippocampal complex.

What appears to have been one of the first studies of the effects of forebrain lesions in birds on a discrimination task was carried out by Layman in 1936 using chickens. Although intending to destroy only the cortical areas, the actual lesions were much more extensive and included hippocampal, parahippocampal, and dorsolateral corticoid tissue, varying amounts of anterior or posterior hyperstriatal tissue, and, variously, parts of the neostriatum, archistriatum, and paleostriatum. The birds were trained to discriminate between a circle and a triangle, which were presented simultaneously in a modified Yerkes-Watson discrimination box, and many of the lesioned chickens learned the task successfully, and as readily as the normal chickens. However, in those birds that were impaired, anterior hyperstriatal lesions tended to have a greater effect than posterior lesions, and extensive lesions caused greater impairment than smaller lesions. From these results, Layman concluded that cortical tissue was not essential for the formation of a visual pattern discrimination but that there were a number of anterior striatal areas which, if destoyed together, would prevent the formation of visual pattern habits, whereas when damaged separately they would not. He also found that, if the cortical lesions were sufficiently large, a visual learning deficit did occur, but he attributed this to "a lowering of the general intelligence of the subject" rather than to a visual impairment (Layman, 1936, p.28).

Reynolds and Limpo (1965) trained five pigeons in a single key chamber on a multiple fixed-interval (FI) 4 mins - fixed ratio (FR) 55 schedule of reinforcement. The first component occurred in the presence of a red light and the second in the presence of a

green light, and the two components were presented alternately. The pigeons were trained until they reached a stable level of performance, and were then operated on. Using vacuum aspiration, HA, most of the dorsomedial region of the hyperstriatum, and the hippocampal area were removed in three of the pigeons and their postoperative performance was compared with that of the other two, sham-operated, pigeons. It was found that the behaviour of all five pigeons on the FR55 component did not change postoperatively, in that a high sustained rate of responding was maintained by both groups, but also the lesioned pigeons occasionally paused at the beginning of this part of the schedule, i.e., following reinforcement on the fixed interval component, although they had not shown this behaviour preoperatively. These pigeons would also suddenly stop responding on several occasions, and for varying periods of time, during the high response rate phase of the fixed interval component, but at the beginning of this component, following reinforcement at the end of the fixed ratio component, they no longer showed the typical pause, it being consistently absent or shorter than normal.

Despite the aberrant pausing behaviour at the beginning of both components of the schedule, Reynolds and Limpo have argued that the behavioural changes were probably not due to any sort of sensory deficit, since the pigeons nevertheless showed a detectable reduction in their overall response rates when the keylight changed from green to red, signalling a change from the fixed ratio to the fixed interval component, and a correspondin increase when the keylight changed from red to green. Thus, they suggested initially that the lesions may have disinhibited responding, but because of the inappropriate pausing behaviour during the fixed interval schedule, they concluded that the effect could not be simply a disinhibition of responding. In fact, the periodic pausing at the beginning of the fixed ratio schedule, and the reduced or absent pausing at the beginning of the fixed interval schedule suggests instead the possibility of some sort of successive discrimination deficit.

An experiment of considerable interest and importance was carried out by Stettner and Schultz (1967). Three groups of Bobwhite quail (Colinus virginianus) were used, an unoperated control group, a sham-operated control group, and a group which had received lesions, produced by subpial aspiration, to the Wulst and the hippocampal, parahippocampal, and dorsolateral corticoid areas. The quail were trained to a 90% correct criterion on a simultaneous pattern discrimination presented in a two-key operant chamber, the stimuli consisting of horizontal or vertical stripes, and subsequently were trained to a criterion of 80% correct on each of 25 serial reversals of the discrimination. All three groups learned the original discrimination in approximately the same number of trials and with the same number of errors, but the lesioned group was impaired on reversal learning. Since the lesions did not affect acquisition performance, and frequency and latency of pecking was not affected, it was assumed that these birds' sensory and motor abilities, and their motivational states, were also unaffected. Instead, their reversal deficit was found to be due to marked perseverative responding to the previously correct stimulus in the early stages of each reversal, although a subsequent analysis of these data (Stettner, 1974) showed that by far the greater proportion of the deficit was due to exaggerated position responding after responding to the previously rewarded stimulus had been abandoned. Also of interest is the further observation that, in the lesioned animals almost total removal of the Wulst had been achieved, but the extent of the damage to the posterior cortical tissue, which included the hippocampal and parahippocampal areas, was quite variable. Two birds that had lost almost all of this cortex were more impaired than the other two in this group, one of which had virtually no damage to the posterior cortical tissue and the other retained more than half of the cortical tissue in this area, and therefore Stettner and Schultz concluded that the extent of the deficit was related to the extent of the cortical damage.

In 1969, Macphail reported three experiments involving pigeons with anterior hyperstriatal lesions, which were produced by means of a scalpel blade and were therefore somewhat variable in extent. In the first experiment, which was the acquisition and reversal of a simultaneous brightness discrimination in a Grice box, none of the six pigeons in the hyperstriatal group was impaired on the acquisition of the task, and five of the pigeons also learned the reversal as quickly as the unoperated control birds. However, the sixth pigeon in the experimental group, which had rather more extensive lesions and included hippocampal damage, took almost twice as many trials and made approximately twice the number of perseverative errors to criterion on reversal as the other pigeons. The second experiment investigated extinction behaviour in a simple runway, and it was found that the five hyperstriatal pigeons took fewer trials to extinction than the control group, but that the pigeon with the more extensive lesions took noticeably more trials than the control group. Finally, the third experiment, using the same runway apparatus as in experiment 2, was a passive avoidance task, and it was found that, on the whole, five of the hyperstriatal pigeons tended to show longer response latencies than the control group, but that the pigeon that also had hippocampal damage showed very short latencies. Thus, those pigeons in which the lesions were restricted to the anterior hyperstriatum responded as well as normal pigeons on the reversal task, and tended to respond more efficiently on the extinction and passive avoidance tasks, thereby showing normal or slightly better than normal ability to inhibit responses in those tasks that require it. On the other hand, the anterior hyperstriatal pigeon that had also sustained damage to the hippocampus did not show such efficient inhibitory behaviour, but instead was impaired in its ability to withhold responses.

The results of these three experiments, then, suggest that the deficits that were found to occur on the reversal, but not the acquisition, of a simultaneous discrimination, simple extinction behaviour, and performance on a passive avoidance task, were primarily

due to hippocampal and parahippocampal involvement, rather than to hyperstriatal damage, since those pigeons in which the lesions were restricted to the hyperstriatal region, or Wulst, did not show these deficits. The results obtained by Stettner and Schultz (1967) provide support for this suggestion, at least as far as the reversal deficit is concerned, and further support can be found in the work of Benowitz (1972), Benowitz and Lee-Teng (1973), and Lee-Teng and Sherman (1969).

Benowitz (1972), using a one-trial passive taste-avoidance task in young chicks, found that limited hyperstriatal lesions, which included HD, HA, IHA, and hippocampal and parahippocampal tissue, impaired the post-operative acquisition of the task, whereas more extensive hyperstriatal lesions, which in addition included parts of the HV and neostriatum, also impaired the postoperative retention and relearning of the preoperatively acquired task. In comparison, frontal forebrain ablations, which included parts of the Wulst, neostriatum, and paleostriatum augmentatum, impaired only the postoperative retention and relearning of the task without affecting post-operative acquisition. Although recognising that the inclusion of several morphologically distinct regions complicated the interpretation of the effects of the lesions, Benowitz proposed that the postoperative acquisition deficit in the limited hyperstriatal group could be due to hippocampal damage, since similar effects in dorsomedially ablated chicks, in which mainly the hippocampal and parahippocampal areas were damaged, had previously been reported by Lee-Teng and Sherman (1969).

Additional evidence implicating the hippocampal complex in a reversal deficit comes from an experiment by Benowitz and Lee-Teng (1973) in which, again using young chicks, they investigated the effects of several types of forebrain lesions on the acquisition and reversal of a simultaneous shape discrimination. The stimuli were a horizontal bar and a vertical bar, and each correct response was reinforced by a 5 secs flow of warm air, at 95^{o}F, the temperature of the experimental chamber otherwise

being maintained at 54°F. Acquisition training was continued until the chicks reached the criterion of 12 consecutive correct responses. Reversal learning then began on the next session and all chicks were run to the same criterion as before, or for a total of fourteen 18 min sessions. The chicks with dorsomedial ablations, which included most of the Wulst and hippocampal and parahippocampal areas, were not impaired on acquisition, but took significantly more trials on reversal than either an unoperated control group, a frontal ablation group, which received damage to the olfactory bulbs, the medial septal nucleus, and parts of the Wulst, neostriatum, and paleostriatum augmentatum, or a posterolateral ablation group in which the archistriatum, parts of the caudal neostriatum, and the adjacent periamygdalar cortex were lesioned. In comparison, neither the frontal nor the posterolateral group was impaired on either acquisition or reversal, although the frontal group achieved, on average, significantly fewer trials per session than the other three groups during reversal, and the early stages of the acquisition curve of the posterolateral group was significantly depressed compared with the other groups. On the grounds that the frontal group, which included hyperstriatal damage, was not impaired on either phase of the discrimination task, Benowitz and Lee-Teng concluded that the reversal deficit of the dorsomedial group, which also received hyperstriatal damage, was most likely due to the ablation of the other structures, which included primarily the hippocampal and parahippocampal areas. In relation to these findings on the acquisition of this task, it will be recalled that Layman (1936) concluded from the results of his experiments that cortical tissue was not essential for the formation of a visual pattern discrimination, but that there were a number of anterior striatal areas which, if lesioned together, would impair the acquisition of a pattern discrimination, although they would not if lesioned separately. Finally, since the hyperstriatal lesions in the experiment by Reynolds and Limpo (1965) included hippocampal and parahippocampal damage, it is possible that these cortical regions were also involved

in the deficits that occurred on the two schedules of reinforcement.

Therefore it is tentatively proposed that lesions of the hippocampal complex in birds affect responding on certain schedules of reinforcement, impair the reversal, but not the acquisition, of a simultaneous discrimination task, and retard extinction and the performance of a passive avoidance task. Of particular interest here is the observation that these deficits are very similar to those that have been found to occur following hippocampal lesions in mammals (see p.45 et seq for a review of the relevant experiments.)

b) Lesions that spare the hippocampal complex

The experiments reported by Zeigler (1963a) were particularly influential in the study of avian forebrain function since they were the first to demonstrate an effect of lesions, confined primarily to the hyperstriatal complex,on a discrimination task. Using a single-key operant chamber and an FI 40 secs schedule of reinforcement, and therefore a successive go, no-go discrimination task, he trained three groups of pigeons on a brightness task, or on a pattern task in which they had to discriminate between a triangle and a circle. Some of the pigeons were trained to criterion preoperatively and then retrained postoperatively (the retention condition), while the others received only postoperative training (the acquisition condition). The three groups were a sham-operated control group, a hyperstriatal group in which the Wulst, HV, and small amounts of corticoid tissue, but excluding the hippocampal and parahippocampal areas, were lesioned by subpial aspiration, and a 'mixed' group which, although intended to have neostriatal lesions, also sustained varying amounts of damage to some of the other striatal regions and to the dorsolateral corticoid area. Damage to the hyperstriatum also occurred in a number of cases in this group, but was only slight. The hyperstriatal group were found to be significantly impaired on acquisition or retention and subsequent relearning of the discrimination tasks, although the latter deficit was primarily a

relearning one, the control group also showing an equivalent day 1 retention loss. Furthermore, these pigeons were more severely impaired on the pattern task than on the brightness task. The mixed lesion group gave rise to some mixed data, but on the whole, pigeons in this group could be divided into those which did not show any impairment on either acquisition or relearning compared with the control group, and those which did. The distinguishing feature of the pigeons in this latter division was that they all had paleostriatal damage in addition to lesions in other areas.

A similar experiment was reported by Pritz, Mead, and Northcutt (1970). They also trained pigeons preoperatively on a successive, go, no-go discrimination task, reinforcing correct responses on a FR 5 schedule, and each pigeon learned either a colour, a brightness, or a pattern (+ vs. x) discrimination. The pigeons were trained to criterion and were then operated on. The lesions were produced by vacuum aspiration, a hyperstriatal group receiving Wulst ablations together with minimal damage to the neostriatum and to the hippocampal, parahippocampal, and dorsolateral corticoid areas, and a lesioned control group received damage to the neostriatum and the dorsolateral corticoid area, although one pigeon in this group also had moderate damage to the hippocampal and parahippocampal regions. A sham-operated group was also used, but even these animals received slight damage to the hyperstriatal complex (including HV), the neostriatum, and the dorsolateral corticoid area. No postoperative retention or relearning deficits on any of these three tasks were found for the two control groups, and similarly no deficits occurred for the hyperstriatal group on the colour discrimination. However, they were impaired on retention and relearning of both the brightness discrimination and the pattern discrimination. Thus these two experiments showed clearly, for the first time, that lesions largely restricted to the hyperstriatal region, although including varying amounts of cortical, neostriatal, and paleostriatal tissue, could produce acquisition and relearning deficits on certain types of visual discrimination tasks.

A series of experiments were also carried out by Macphail, between 1971 and 1976, in which the hyperstriatal complex alone was lesioned so as to leave the hippocampal complex intact. In the first report of this series Macphail (1971) described two experiments. The first was a free-operant successive go, no-go colour discrimination in a single-key chamber. Two groups of pigeons, a hyperstriatal lesioned group and a group of unoperated and sham-operated control pigeons, were pretrained to respond to either a red or a green keylight on a variable interval (VI) 60 secs schedule of reinforcement until a stable response rate was established, and then discrimination training began, in which only responses to the green keylight were reinforced. No differences were found to occur between the two groups in pretraining, but during discrimination training the hyperstriatal group, although described as not suffering from a gross impairment of response inhibition, made significantly more responses on the negative trials than the control group in the first few components of each day over the first few days. No differences, however, were found to occur on the positive trials.

These pigeons were then pretrained to peck either green side-key in a three-key chamber, followed by acquisition training on a discrete-trials simultaneous position discrimination, in which both side keys were green and a response to the right-hand key was reinforced, and six daily reversals. No deficits were found on acquisition, but on each of reversals 1 and 2, and on reversals 3-6 combined, the hyperstriatal pigeons made significantly more errors to criterion than the control group.

Macphail (1975a) reported two further experiments, the first of which was the acquisition and five serial reversals of a simultaneous position discrimination. Four groups of pigeons were used: two control groups, one of which was unoperated and the other received neostriatal lesions, and two experimental groups, one of which had received anterior hyperstriatal lesions and the other, posterior hyperstriatal lesions. They were pretrained to peck either side key when lit with either red or green light,

and then during acquisition training half of each group was trained with their initially preferred side positive, and the other half with it negative. On reaching criterion five reversals were given. As before, no differences occurred during pretraining, but in this experiment both hyperstriatal groups were impaired on the acquisition as well as on the reversals, although training with or against side preference had had no effect. In the five reversals the anterior hyperstriatal group made significantly more errors than the posterior group, but this effect appeared to be due to the larger size of the anterior lesions. Further analysis showed that the hyperstriatal pigeons in both groups stopped responding and required free reinforcements significantly more often than the control pigeons.

These pigeons were then trained in the second experiment, which was a successive go, no-go colour discrimination presented as a discrete trials task in a three-key chamber. On positive trials the two side keys were green and remained on for 8 secs or until a total of five responses, distributed in any way, were made, and then food was delivered. On negative trials the two keys were red for 4 secs and responses on them had no effect. Neither hyperstriatal group took significantly more trials to criterion than either control group, but when the numbers of responses in negative trials were determined separately for the preferred and nonpreferred keys (defined solely in terms of percentages of total negative responses) it was found that the two hyperstriatal groups, which themselves did not differ, made a significantly higher percentage of negative responses to the preferred key than either control group. However, in this experiment the hyperstriatal pigeons did not show a greater tendency than the control pigeons to stop responding.

In the third report in this series, Macphail (1976a) described two experiments, both of which consisted of within-day reversals of a simultaneous discrimination, and separate groups of pigeons were used in the two experiments. The first experiment was the pre-operative acquisition of a red-green discrimination, and then 50 serial reversals over

17 days, followed postoperatively by a further 18 reversals over 6 days. The hyperstriatal lesioned pigeons made significantly more perseverative errors and more position responses on each of the three daily reversals compared with the sham-operated control group, and also had a greater tendency to stop responding. The second experiment was a position discrimination, and the procedure was the same as that in experiment 1 except that both side keys were red on each trial. Again, the hyperstriatal pigeons made significantly more perseverative errors in each reversal compared with the control group, and they also had a greater tendency to stop responding.

Finally, Macphail (1976b) reported an experiment in which a group of hyperstriatal lesioned pigeons and a group of unoperated controls were trained postoperatively in a three-key apparatus, first on the acquisition of a simultaneous position discrimination and then on the acquisition and four serial reversals of a simultaneous colour discrimination. In order to resolve the discrepancy in the results of the two earlier experiments on the acquisition of a position discrimination (Macphail, 1971, 1975a), which was believed to be due to different amounts of pretraining, half the pigeons in each group in the present experiment were given minimal pretraining to peck the illuminated side key regardless of its colour (red or green) or position, while the remaining animals were given extended pretraining. Then position discrimination training began, with colour irrelevant, and half the pigeons in each group were trained with their initially preferred side positive, and the other half with it negative. On reaching criterion the pigeons were trained on the acquisition of the red-green discrimination, followed by four reversals.

The results showed that the hyperstriatal pigeons were impaired on the acquisition of the position task following extended, but not minimal, pretraining compared with the control group, but that training against initial preference had no effect. No deficit occurred on the acquisition of the colour discrimination, but the hyperstriatal pigeons were impaired on the reversals, mainly due to increased perseverative responding.

Finally, as in several of the previous experiments, the hyperstriatal group stopped responding significantly more often than the control group over the two stages of this experiment.

In a related experiment Pasternak (1977) investigated the effects of similar hyperstriatal lesions on a delayed matching to sample (MTS) task. Eight pigeons were pretrained in a three-key chamber on a zero-delay MTS: the centre key, which was illuminated with either green or yellow light (the sample stimulus) was switched off by a single peck, the two side keys, one of which was green and the other yellow (the comparison stimuli) were switched on, and a response to the comparison stimulus which matched the colour of the sample was food-reinforced. Delays of 1, 2, 4, and 8 secs were also used, but each increase in the delay was only introduced when the pigeons were performing at 90% correct on the current delay. Finally, they were pretrained on a simultaneous MTS in which the sample stimulus remained on while the comparison stimuli were presented. This was followed by extensive training and retention trials on a mixed delayed MTS in which the delays were presented in a random sequence. Six pigeons then received hyperstriatal lesions which involved HA, IHA, HISm, HD, and HV, and two pigeons, which served as operated controls, received minor damage to HA and HISm. Postoperatively the pigeons were retested on the mixed delayed MTS task and the performance of the two control pigeons was found to be unaffected by surgery. In comparison, the anterior hyperstriatal pigeons were grossly impaired and their performance on each of the delays fell to chance level, due to an almost total preference for the yellow comparison stimulus. Extensive retraining substantially improved their performance on the simultaneous and zero-delay conditions, but on the whole their performance on the other delays remained around chance level. Pasternak assumed that, because the hyperstriatal pigeons were clearly able to discriminate between the colours (in order to be able to respond consistently to yellow), the deficit was due

the conditional nature of the task (the changing relation of the side-key stimuli to reinforcement), and that it was possibly related to the visual reversal deficits that occur after hyperstriatal lesions, since the colour preference responses in the MTS task appeared to be similar to the perseverative errors that occur in reversal learning.

In the experiments of Macphail (1969, 1971, 1975a, 1976a, and 1976b) and Pasternak (1977) the experimental groups received bilateral lesions which extended, variously, from A14.00 to A9.00 according to the stereotaxic coordinates of Karten and Hodos (1967), and included damage to HA, IHA, HISm, HD, and HV.

The effects of corresponding hyperstriatal lesions were also investigated in an experiment by Hodos, Karten, and Bonbright (1973). They trained unoperated pigeons on four simultaneous discrimination tasks (one brightness and three pattern problems), presented concurrently in a two-key chamber until they reached criterion on each of the problems. They were then operated on and retrained on the discrimination tasks. When the hyperstriatal lesioned pigeons were compared with an operated control group, it was found that they did not show any postoperative retention or relearning deficits on any of the four visual discrimination problems. However, Pasternak and Hodos (1977) investigated the effects of hyperstriatal lesions on visual intensity threshold differences using a successive discrimination technique in a three-key operant chamber. The pigeons were trained preoperatively using a number of stimulus pairs until a stable level of performance was attained on each pair and then they were operated on. The hyperstriatal lesions in the experimental group extended from A14.00 to A8.50 and included HA, IHA, HISm, HD, and HV, and two control pigeons received lesions mainly of the caudal neostriatum, but including parts of the dorsolateral corticoid area, together with some minor damage to HV. The pigeons were then retrained on the discrimination task and it was found that the hyperstriatal lesioned pigeons showed an immediate postoperative increase in their thresholds which was equivalent to a 19% - 49% loss

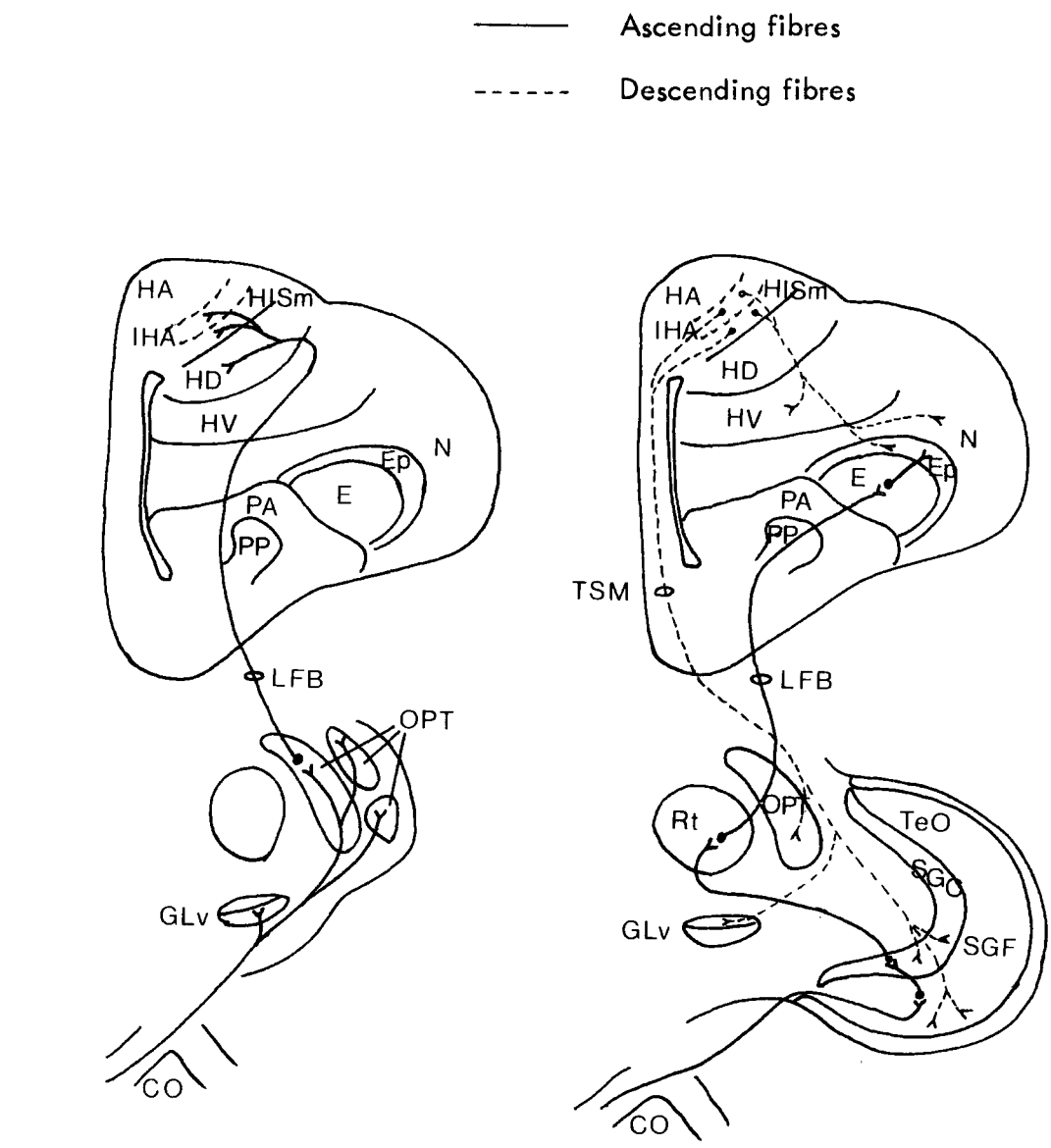
of sensory capacity, although five out of the six pigeons showed improvement with retraining. In comparison, neither of the control pigeons showed any postoperative changes in their thresholds.

In summary, therefore, these various experiments showed that pigeons with lesions restricted to the hyperstriatal complex were impaired on the acquisition or the post-operative relearning of a successive brightness or pattern discrimination, but not a colour discrimination, unless it was presented as a free-operant task. They were also unimpaired on the acquisition of a simultaneous position, colour, brightness, or pattern discrimination, unless given extensive pretraining with position and colour irrelevant, in which case the acquisition of a simultaneous position discrimination was impaired. However, hyperstriatal lesioned pigeons were impaired on the reversal of a simultaneous position or colour discrimination, but not a brightness discrimination, and on a delayed matching to sample task. Finally, their brightness difference thresholds were increased, and in most of Macphail's experiments they were found to stop responding significantly more frequently than the control pigeons. Some of these effects have also been found in hippocampal lesioned mammals (see p. 45 et seq for a review of the relevant experiments).

Anatomical and electrophysiological studies of the hyperstriatal complex

During the 1960's several investigators began to study the afferent projections from the retina in birds. Using the Fink-Heimer silver methods for degenerating axons and terminals (Fink and Heimer, 1967), Karten and Nauta (1968) confirmed the earlier finding, by Cowan et al (1961), of a projection from the retina to the contralateral dorsolateral anterior thalamic complex, but found that it was considerably more extensive than had previously been supposed. Because of the size of this projection and the number of cell groups that were involved, Karten et al (1973) called this thalamic complex the nucleus opticus principalis thalami (OPT). Karten and Nauta (1968) and Karten et al (1973

were also able to show that the efferent projections from OPT entered the ipsilateral and, via the dorsal supraoptic decussation, the contralateral lateral forebrain bundles (LFB) and finally terminated mainly in the intercalated nucleus of the accessory hyperstriatum (IHA), but also in the hyperstriatum intercalatus suprema (HISm) and the dorsal hyperstriatum (HD). This pathway is known as the thalamofugal pathway (see Figure 3A), and the hyperstriatal region in which the OPT fibres terminate has since been designated the 'visual Wulst' (Karten et al, 1973). Efferent fibres from the visual Wulst have been found to project on to the ipsilateral ventral hyperstriatum (HV), the lateral neostriatum (N), and the periectostriatal belt (Ep), a cytologically differentiated band surrounding the ectostriatum (E) (Karten and Hodos, 1970; Karten et al, 1973). In addition to these projections other efferent fibres from the visual Wulst have been found to form a major component of the septomesencephalic tract (TSM), the rostromedial division, which descends without termination in the hippocampus, the preoptic nucleus, or the hypothalamus, to distribute its fibres bilaterally, via the dorsal supraoptic decussation, to the ventral portion of the lateral geniculate nucleus (GLv), a small region of OPT, the pretectal nuclei, and the optic tectum (TeO) (Hunt and Webster, 1972; Karten et al, 1973). Electrophysiological studies of the hyperstriatum have been carried out by Revzin (1969) who found single units in IHA which had small circular or relatively elongated receptive fields, ranging from 0.5° to 4° and rarely exceeding 10°, and which showed good topographic organisation and were organised in columns. Very recently, Pettigrew (1979) has reported that electrophysiological recordings from single neurones in the visual Wulst of owls have shown that many of the neurones possess the receptive field properties of single cells in areas 17 and 18 in the cat and the monkey. Cells were found which had properties very similar to those described by Hubel and Wiesel (1962, 1965, 1968) as simple, complex, and hypercomplex I cells. In the owl the majority of the simple cells



A Thalamofugal pathway

B Tectofugal pathway

Figure 3. Schematic illustration of the two principal visual pathways from the retina to the telencephalon and the efferent projections from the visual Wulst in the pigeon. For abbreviations see text.

were binocular, and all of the complex and hypercomplex I cells could be binocularly driven.

Other studies had previously established the existence of another visual projection system in birds, which has been called the tectofugal pathway (see Figure 3B). Although the projection from the retina to the contralateral optic tectum, and from there to the nucleus rotundus of the thalamus (Rt) had been known for a long time (see Ariëns Kappers et al, 1936), there had been the suggestion, based on studies of normal histological material, that the nucleus rotundus also received somatosensory fibres, particularly from the gracile and cuneate nuclei. However, there were other early reports, based on degeneration studies, in which these somatosensory projections had not been found, and this was only recently properly confirmed by Karten (1963-1965; see Karten 1969) and Karten and Revzin (1966). Thus, the nucleus rotundus was found to receive only a massive projection indirectly from the retina, via the optic tectum. Then, using electrophysiological (Revzin and Karten, 1966/67) and anatomical (Karten and Hodos, 1970) techniques, it was found that the nucleus rotundus also sent a massive projection, which formed the lateral part of the lateral forebrain bundle, through parts of the paleostriatum primitivum (PP) and augmentatum (PA) to terminate in a topographic manner in the central core of the ectostriatum. It now appears that there are further projections from the central core of the ectostriatum to the periectostriatal belt (Karten et al, 1973), although this had previously been in doubt (Karten and Hodos, 1970), as well as to the intermediate neostriatum and to a laminated population of cells in the dorsolateral surface of the hemisphere (Karten, 1969; Cohen and Karten, 1974). Electrophysiological studies by Revzin and Karten (1966/67) and Revzin (1970) have shown that single units in all parts of the tectofugal system, in contrast to those in the visual Wulst, have extremely large receptive fields, often in excess of 90°, and are particularly sensitive to movement.

The correspondence between these two visual systems in birds and the visual systems of mammals is quite striking. As well as the anatomical similarities between the avian thalamofugal system and the mammalian geniculostriate system, the electrophysiological characteristics of single units in the multilaminate visual Wulst, together with their columnar arrangements, are remarkably reminiscent of the major properties of single cells in the striate cortex of cats that were first described by Hubel and Wiesel (1962). In addition, there are the recently reported similarities in the orientation - specific and binocular characteristics of single cells between the visual Wulst of owls and the striate cortex of mammals. The tectofugal system, on the other hand, bears many similarities to the mammalian colliculo-thalamo-circumstriate system that was first recognised in the cat by Altman and Carpenter (1961), and subsequently found in other mammals (Diamond and Hall, 1969; Schneider, 1969) and in the turtle (Hall and Ebner, 1970).

The anatomical studies of Karten et al (1973) have further shown that the dorsomedial anterior thalamic complex (DMA) projects to the Wulst, independently of the telencephalic projections from OPT, and the fibres terminate in the medial region of HD and in the immediately adjacent ventromedial portion of the Wulst, adjoining the hippocampal area. Since the efferent fibres from DMA ascend in close association with the medial forebrain bundle (MFB), Karten et al have suggested that the DMA and its projection field in the Wulst, adjacent to the hippocampal formation, may be comparable to the mammalian anterior thalamic complex and its telencephalic target, the cingulate cortex. In addition, Karten (1971) has shown that a region of the hyperstriatal complex, anterior to the visual Wulst, gives rise to fibres that form the basal branch of the TSM. It distributes fibres to several nuclear groups including the red nucleus and then continues caudally into the midbrain and onto the ventral surface of the brainstem. Other fibres terminate in the reticular formation and in the pontine,

cuneate, and gracile nuclei, and the main bundle of fibres decussates at the bulbo-spinal junction and descends in the contralateral dorsal funiculus of the spinal cord. In many respects the basal branch of the TSM and its various interconnections closely resembles a component of the mammalian pyramidal system.

Thus, it is clear that the hyperstriatal complex consists of at least three distinct regions that differ cytoarchitectonically and in their fibre connections with other regions of the brain (Karten et al, 1973). The anterior region of the Wulst is the source of a projection system that is very similar to part of the mammalian pyramidal tract, and therefore may be comparable to the equivalent cortical region in the mammalian brain. Immediately posterior to this is the visual Wulst which bears many similarities to the mammalian striate cortex. Finally, there is a ventromedial region of the Wulst (Karten et al, 1973, pp. 273 and 274; however, on p. 262 they refer to this area as the dorsomedial region of the Wulst) which, because of its location next to the hippocampal area, and its afferent connections with the thalamus, has been compared with the mammalian cingulate cortex.

Functional aspects of the hyperstriatal complex

Altogether, only a relatively small number of experiments on the behavioural effects of hyperstriatal lesions have been reported, and only a rather limited number of behavioural tasks have been investigated. In some of these experiments the lesions included damage to the hippocampal area, and in a review presented earlier in this chapter it was suggested that the deficits that occurred in these experiments were due to the hippocampal damage, particularly as the same deficits have been reported in hippocampal lesioned mammals. However, a review of other experiments on the effects of hyperstriatal lesions revealed that one of the deficits following combined hyperstriatal and hippocampal damage, a reversal learning deficit, also occurred in pigeons in which the lesions had been restricted to the hyperstriatal complex, thereby leaving the

hippocampal area intact. Clearly, therefore, hyperstriatal lesions alone can cause a reversal learning deficit; but although it is more parsimonious to attribute the reversal learning deficit solely to the hyperstriatal damage, it does not follow that a similar deficit would not occur in birds in which only the hippocampal area was lesioned. One of the other effects of 'restricted' hyperstriatal lesions was the impairment of a successive discrimination task, another deficit that is also commonly found in hippocampal rats. Again, it is possible that hippocampal lesions alone in pigeons would give rise to a similar deficit, but until such data are available this must remain a speculation.

Other deficits were found to occur which do not have their parallels in the mammalian hippocampal literature. First, Pasternak (1977) found that pigeons with restricted hyperstriatal lesions were impaired on a delayed matching to sample task and, as far as the writer is aware, the only experiment that has been reported in which hippocampal lesioned mammals have been tested on the same type of task is that by Correll and Scoville (1965), who found that hippocampal lesions in monkeys did not impair their matching performance with delays up to 5 secs.

Secondly, in a number of his experiments, Macphail has reported that pigeons with restricted hyperstriatal lesions (but not the one subject [Macphail, 1969] in which the lesion involved hippocampal tissue) stopped responding significantly more often than the control animals, thereby showing what could be described as an increase in response inhibition. It will also be recalled that Reynolds and Limpo (1965) observed abnormal pausing in combined hyperstriatal and hippocampal lesioned pigeons on an FI 55 secs schedule of reinforcement (although it should be noted that these pigeons did not show the usual post-reinforcement pause at the beginning of the FI component).

Macphail (1975b) reviewed the evidence in relation to the response-inhibition hypothesis of hyperstriatal function and concluded that, although this hypothesis could account for much of the hyperstriatal data, the findings of halting behaviour and

abnormal pausing created some difficulties. Subsequently, however, Macphail (1976b) obtained evidence that supported the response-inhibition hypothesis and contradicted a response-shift hypothesis (see Olton, 1972a). It has also been suggested that hippocampal lesions in mammals cause a loss of response inhibition (Kimble and Kimble, 1965; McCleary, 1966; Altman, Brunner, and Bayer, 1973; see also Douglas, 1967), but evidence is gathering which shows that these animals need not suffer from an inability to withhold responses (Winocur and Salzen, 1968; Gaffan, 1972; Stevens, 1973b; Nadel, O'Keefe, and Black, 1975; Winocur and Black, 1978).

Cingulate cortex lesions have been reported to increase response inhibition in several experimental situations, and particularly in avoidance tasks (see Isaacson, 1974). Thus, McCleary found that cats with cingulate lesions were impaired on a two-way active avoidance task, but not on a passive avoidance situation, although Lubar (1964) reported that similar animals were not impaired on one-way active avoidance learning, and were superior to normal cats on a passive avoidance task. But Brutkowski and Mempel (1961) found that dogs with cingulate lesions were impaired in their ability to withhold responses to a nonrewarded cue, which they interpreted as due to a loss of inhibition. Furthermore, Barker (1967) reported that rats with anterior cingulate lesions were impaired in a response alternation task in an operant chamber. It appears, however, that these differences can be explained, at least partly, in terms of which region of the cingulate cortex is damaged. Isaacson (1974) states that anterior cingulate lesions are more likely to produce impaired response suppression or inhibition than are posterior lesions.

Since Karten et al (1973) have suggested that, on account of its connections with the anterior thalamic complex, the ventromedial Wulst may be comparable to the mammalian cingulate cortex, it is tempting to suggest that the halting phenomenon and the loss of response inhibition reported by Macphail, and the pausing found by

Reynolds and Limpo, were due to ventromedial Wulst damage.

Possibly related to this halting or pausing behaviour is the reduction in activity and responsiveness in birds with hyperstriatal damage that has been noted by a number of observers. Rogers (1922) and Tuge and Shima (1959) both found a marked hypokinaesia in their lesioned birds, although varying degrees of recovery occurred in time. Zeigler (1963b) reported a postoperative period of decreased locomotor activity in a group of hyperstriatal lesioned pigeons, so that, when first placed in an activity cage under constant conditions, their level of activity was significantly lower than that of normal pigeons. However, he also found that they did not show the decline in responsiveness over time that occurs in normal pigeons. Nevertheless, Zeigler, too, found that these effects were transient and that they disappeared within 3-6 months. Further evidence for a reduction in responsiveness to external stimuli was obtained by Cohen (1967), who reported that, following hyperstriatal lesions, pigeons were less responsive to flashes of light and slowly moving stimuli compared with normal pigeons, and that intense visual or tactile stimuli were required to elicit escape behaviour, a finding that had also been reported by Rogers (1922). The findings of Karten et al (1973) of the similarities between the mammalian pyramidal tract and the basal branch of the TSM that projects from the anterior hyperstriatal region have already been referred to, but earlier, based on the results of her anatomical studies of hyperstriatal efferent fibres, Adamo (1967) had also proposed that this projection could be considered to be a hyperstriato-ponto-cerebellar feedback system similar to the mammalian cortico-ponto-cerebellar system, and she therefore suggested that part of the Wulst was important in the modulation and control of motor activity. Subsequently, on the basis of his earlier behavioural studies, Macphail (1969) suggested the existence of a facilitatory mechanism in the anterior hyperstriatal region. More recently, a similar, although rather more detailed, proposal has been made by Salzen and Parker (1975),

and it would seem possible, therefore, that a disruption of this facilitatory mechanism could equally well lead to abnormal breaks in responding.

Finally, Pasternak and Hodos (1977) found that restricted hyperstriatal lesions in pigeons significantly increased their brightness difference thresholds, and this finding has yet to be reported in hippocampal mammals. Instead, Hodos et al (1973) and Pasternak and Hodos (1977) have noted that both this effect and some of the other behavioural effects of hyperstriatal lesions are similar to those produced by striate cortex lesions in mammals. For example, Lashley (1930) found that extensive striate cortex ablations in rats did not impair a preoperatively learned brightness habit, but their difference thresholds were higher than those of normal rats. However, total striate cortex removal did produce a postoperative deficit, although the animals were able to relearn the task in about the same number of trials as they had originally taken. Smith (1937) showed that destriate cats were unimpaired on a preoperatively learned brightness discrimination if the only source of illumination in the apparatus was that provided by the stimuli themselves, but that they were impaired if the stimuli were presented in a low level of general illumination. And Schilder, Pasik,and Pasik (1971) have shown that destriate monkeys can learn a brightness discrimination even when the stimuli are equated for total luminous flux, while Butter (1974) and Pasik and Pasik (1971) have reported that monkeys with either lateral or total striate ablations can learn a colour discrimination. Although earlier reports (reviewed by Weiskrantz, 1961) suggested that a loss of pattern vision occurred as a result of striate cortex ablation, more recently Weiskrantz (1963) showed that a young rhesus monkey with almost total striate cortex removal was able, with extended training, to learn a discrimination between patterns that differed in total contour but were equal in total luminous flux. Subsequently, similar findings were obtained for the rat (Cowey and Weiskrantz, 1971; Mize, Wetzel, and Thompson, 1971) and the cat (Wetzel, 1969; Dalby, Meyer, and

Meyer, 1970). In lower mammals, Hall and Diamond (1968) have shown that removal of the visual cortex in the hedgehog produces pattern deficits, although Snyder and Diamond (1968) have found that similar lesions in another primitive mammal, the tree shrew (Tupaia glis), do not affect the acquisition of a simultaneous pattern discrimination.

According to Hodos et al (1973), in their attempt to explain this discrepancy, Snyder and Diamond (1968) have suggested as one possibility "that the visual system of tree shrews may bear a greater similarity to that of the ancestral mammal-like reptiles" (Hodos et al, 1973, p. 465). Hodos et al then suggest that the similarity between their findings in pigeons and those of Snyder and Diamond in tree shrews makes this possibility more likely. However, it is important to note that the hedgehog, an insectivore, is a more primitive mammal than the tree shrew, which can be regarded as representing a form that is transitional between insectivores and lower primates (Schneider, 1969), and evidence presented by Diamond and Hall (1969) and Schneider (1969) suggests a more probable explanation. In the hedgehog visual fibres from the retina project via two pathways to a large area of the posterior neocortex, which consists of two cytoarchitectonically distinct regions, a visual core area and the visual belt, corresponding, respectively, to the striate cortex and the circumstriate cortex of higher mammals. One set of fibres from the retina projects to the lateral geniculate nucleus (LGN), while the other projects to the superior colliculus and then to the lateroposterior nucleus of the thalamus (LPN). The LGN and the LPN then send overlapping projections to the visual core and the visual belt (Figure 4A). In contrast, in the tree shrew fibres from the LGN and from the LPN (which has become more like a primate pulvinar) project to, respectively, the striate cortex and the circumstriate cortex without overlapping (Figure 4B). Thus, in the hedgehog the two visual systems are not completely independent, whereas they are in the tree shrew and in the higher mammals.

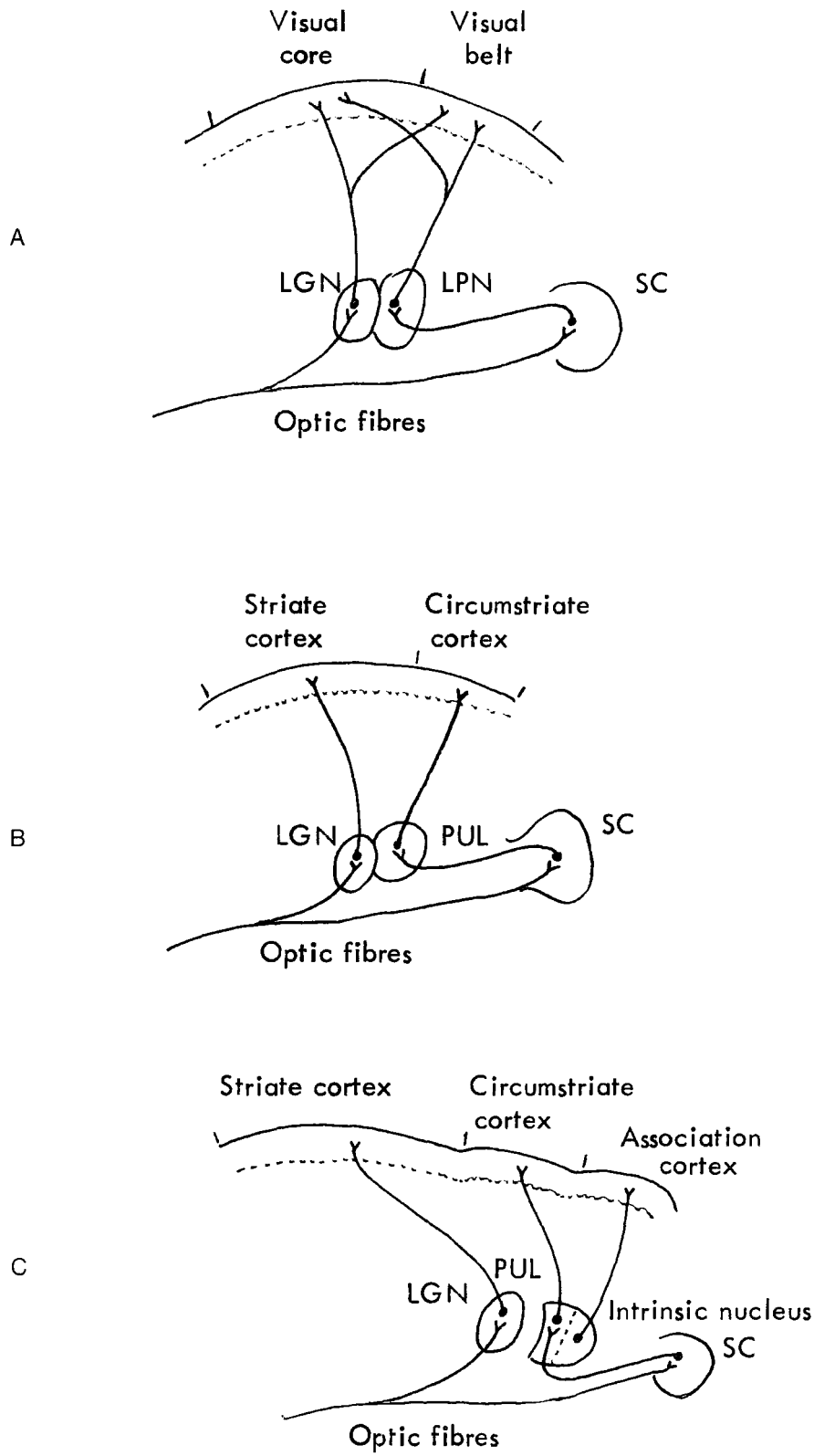


Figure 4. Schematic representation of the evolution of the thalamic and cortical visual areas. A) represents the early mammals, e.g. the hedgehog. B) represents an intermediate stage, e.g. the tree shrew. C) represents the higher mammals, e.g. a monkey. (After Diamond and Hall, 1969). Abbreviations: LGN = lateral geniculate nucleus; LPN = lateroposterior nucleus of the thalamus; PUL = pulvinar; SC = superior colliculus.

There is now considerable evidence from lesion studies which shows that the two visual systems have separate functions, since lesions of the striate cortex have different effects on performance on visual discrimination tasks from those produced by lesions of the superior colliculus. For example, Diamond and Hall (1969) reported that removal of the striate cortex in the tree shrew had no effect on either the acquisition or the reversal of a simple pattern discrimination, but resulted in a complete inability to discriminate between the same stimuli when each was surrounded by a circle. In contrast, removal of the circumstriate cortex impaired both the acquisition and serial reversals of a simultaneous brightness discrimination. Also, Butter (1979) has shown that partial striate cortex lesions in monkeys moderately impaired their postoperative retention and relearning of a simultaneous discrimination involving masked patterns, but their performance on a simple task in which the stimulus and the response were separated was not affected. On the other hand, monkeys with superior collicular lesions were unimpaired on the pattern discrimination, but they were impaired on the stimulus-response separation task. It must therefore be the case that combined lesions of the striate cortex and the circumstriate cortex, or the superior colliculi, would produce a considerably greater impairment on visual discrimination tasks than would lesions of either area alone. And since the two visual systems in the hedgehog are not anatomically separate but instead send overlapping projections to the cortex, it seems most likely that lesions of the so-called visual core, or of the visual belt, in the hedgehog would damage both visual systems, thereby resulting in a greater deficit.

As the review of anatomical and electrophysiological studies presented above has shown, there are two visual systems in the avian brain, and they are very similar in many respects to the visual systems of mammals. Furthermore, they are anatomically separate, unlike those of the hedgehog. It would appear, then, that part of the hyperstriatal complex, the so-called visual Wulst, is comparable to the mammalian

striate cortex, and Hodos and Bonbright (1974) have even suggested that the avian and mammalian visual systems may be homologous. However, Pettigrew (1979) believes that the similarities in binocular visual processing between the visual Wulst of the owl and the mammalian striate cortex are an example of parallel or convergent evolution. It perhaps should not be surprising, therefore, that some of the effects of hyperstriatal lesions on visual discrimination tasks are similar to the behavioural effects of striate cortex lesions.

Earlier in this chapter it was stated that all of the effects that have so far been reported to occur in birds with combined hyperstriatal and hippocampal lesions, namely impaired responding on certain schedules of reinforcement, reversal learning, extinction behaviour, and passive avoidance learning, and some of the effects in birds with lesions confined to the hyperstriatal complex, i.e., deficits on reversal learning and on successive, go, no-go discriminations, are also found in mammals with hippocampal lesions. Furthermore, it was suggested that in those birds with combined hyperstriatal and hippocampal lesions the behavioural effects could have been due to the hippocampal damage. Where similar deficits occurred as a result of hyperstriatal lesions alone it was argued that they might also have occurred in birds with lesions restricted to the hippocampal complex. Macphail (1975a) has made the point that hippocampal damage in birds is not necessary for a reversal deficit to occur, but it is proposed here that similar behavioural effects may occur following lesions in different areas of the avian forebrain. Certainly there is ample evidence that this is the case in the mammalian brain. In fact, Douglas (1967), in his discussion of the specificity of the lesion effects to the hippocampus in mammals, has said that, because there are many reasons why an animal may be impaired on a task, it is inevitable that lesions of even functionally independent brain structures will produce similar behavioural effects.

In the mammalian brain, lesions of the frontal lobe give rise to a number of behavioural changes that are very similar to those produced by hippocampal lesions. For example, Jacobsen and Nissen (1937) found that frontal lesioned monkeys were impaired on a delayed-alternation task, and subsequently deficits were found to occur on this task following hippocampal lesions (Orbach, Milner, and Rasmussen, 1960; Rosvold and Szwarcbart, 1964) and cingulate cortex lesions (Pribram, Wilson, and Connors, 1962). Warren (1964) found that cats with frontal lesions were impaired on the reversal of a spatial discrimination, and Mishkin (1964) reported that frontal lesioned monkeys were impaired on both spatial and object reversals, but in neither case did these animals show a deficit on the acquisition of the discrimination. Frontal lesioned monkeys have also been found to be impaired on auditory (Weiskrantz and Mishkin, 1958; Battig, Rosvold, and Mishkin, 1962) and visual colour and pattern (Battig et al, 1962) successive go, no-go discrimination tasks, unless they are given special training on the no-go trials, in which case they can perform as well as normal monkeys (Pribram and Mishkin, 1956). Further evidence to show that hippocampal and frontal lesions can have similar effects comes from an experiment by Pribram (1961), in which he found that frontal lesioned monkeys were impaired in their performance on an alternating FR 40 - FI 4 mins schedule of reinforcement, a finding which is not unlike that of Reynolds and Limpo (1965) in pigeons with combined hyperstriatal and hippocampal lesions. However, although delayed matching to sample deficits have not been reported in hippocampal lesioned mammals, Mishkin, Prockop, and Rosvold (1962) and Buffery (1967) found that frontal lesioned monkeys were impaired on such a task, and it will be recalled that Pasternak (1977) reported a similar impairment in hyperstriatal lesioned pigeons.

The behaviour of frontal lesioned monkeys has often been described as perseverative, and the same has been said of mammals with hippocampal lesions (see Douglas, 1967)

and of birds with lesions confined to the hyperstriatal complex, or with combined hyperstriatal and hippocampal lesions (see above). Also, various proposals have been made that changes in attention accompany lesions of the frontal cortex (e.g., Buffery, 1967), the hippocampus in mammals (Douglas and Pribram, 1966, Silveira and Kimble, 1968), and the hyperstriatal complex (Hodos et al, 1973; Stettner, 1974; Macphail, 1975; Salzen and Parker, 1975). It can be seen, therefore, that there are a number of similarities not only between birds with hyperstriatal or combined hyperstriatal and hippocampal lesions and hippocampal lesioned mammals, but also between frontal and hippocampal lesioned mammals.

Although he is clearly not suggesting that the hyperstriatal complex and the mammalian hippocampus are homologous, in a review paper Macphail (1975b) has repeatedly compared the effects of lesions in these two areas, suggesting that there is "a family resemblance between deficits brought about by the two types of lesion, and it may well be that the use of situations that have proved sensitive to hippocampal lesions will aid the analysis of hyperstriatal lesions" (p. 159). But it could also be argued that the use of behavioural tasks such as those on which frontal cortex lesioned monkeys have been found to be impaired might be similarly fruitful. However, the anatomical and electrophysiological studies of the hyperstriatal area have clearly shown that it is a complex structure which is likely to be involved in a number of different functions (a point that Macphail [1975b, p. 147] has already made). It was argued above that some of the behavioural effects of hyperstriatal lesions supported the anatomical or electrophysiological evidence which showed there to be a strong resemblance between parts of the hyperstriatal complex and the striate cortex, the cingulate cortex, and part of the pyramidal system of mammals. Since there is a variety of evidence from anatomical, embryological, and histochemical studies (which are reviewed below - see p. 67 et seq) that supports the proposal that the avian

hippocampus and the mammalian hippocampus are homologous, it would seem appropriate, in the absence of behavioural studies involving lesions restricted to the hippocampal complex in birds, to attempt to compare instead the effects of combined hyperstriatal and hippocampal lesions in birds with those of hippocampal lesions in mammals. However, while a wide variety of situations have been used in the study of the mammalian hippocampus, only a rather limited range of behavioural tasks has so far been presented to birds with either restricted hyperstriatal lesions or with combined lesions of the hyperstriatum and the hippocampus.

Behavioural effects of hippocampal lesions in mammals

a) Successive, go, no-go discrimination tasks

It would appear that performance on a successive go, no-go discrimination task is somehow related to performance on a reversal task, since brain-lesioned animals that show a deficit on one of these tasks more often than not show a deficit on the other. Thus, although birds with combined hyperstriatal and hippocampal lesions have yet to be tested on a successive go, no-go task, they have been shown to be impaired on a reversal task (Stettner and Schultz, 1967; Benowitz and Lee-Teng, 1973), and it therefore seemed appropriate to include here a survey of some of the experiments in which hippocampal lesioned mammals have been trained on various successive discrimination tasks.

Successive discrimination deficits have been found in hippocampal rats by Niki (1965) and by Woodruff et al (1973) using a bright light presented only during the positive trials, the lesioned animals making consistently more responses during the negative trials compared with the control animals. It has also been found that cats with hippocampal lesions were impaired on the acquisition of a visual pattern go, no-go task (Buerger, 1969).

In a further experiment, Buerger (1970) trained cats on a go, no-go task in which

the response key was illuminated on positive trials and a 300 Hz tone was presented during negative trials. Each correct response was rewarded with condensed milk, and all inappropriate responses were punished by mild footshock. When trained preoperatively on this task, hippocampal cats did not show any postoperative retention or relearning deficits, but naive hippocampal cats were impaired on postoperative acquisition of the task. In a successive task in which a 1500 Hz tone was presented on positive trials only, and an irrelevant cue light was presented on all trials, Freeman and Kramarcy (1974) found that hippocampal lesioned rats were not impaired. However, when the stimulus conditions were reversed, so that the auditory stimulus was now the irrelevant cue and the cue light signalled the positive trials, the hippocampal rats made significantly fewer responses on the positive trials and significantly more on the negative trials compared with the sham-operated controls. Using the same auditory and visual stimuli, Freeman (1978) found that when one of the cues was presented on positive trials and the other on the negative trials of a successive go, no-go discrimination, hippocampal lesioned rats were impaired. However, when the light and the tone were presented together on the positive trials, neither stimuli occurring during the negative trials, or vice versa, the performance of the lesioned and the control rats did not differ.

Using an auditory cue (160 clicks/min) to signal the negative trials during the acquisition of a go, no-go discrimination, Swanson and Isaacson (1967) reported that hippocampal rats had significantly higher response rates on the negative trials than either sham-operated or cortically lesioned controls, but on the reversal of the task, in which the auditory cue now accompanied only the positive trials, both hippocampal and cortical control rats were impaired in their ability to withhold responses during the negative trials. In a similar task, in which a 2500 Hz tone was presented during positive trials, Schmaltz et al (1973) found that hippocampal lesions in rats made significantly more incorrect responses compared with the control animals. However,

also in a similar auditory go, no-go task, using a 1500 Hz tone, Freeman et al (1973) found that hippocampal rats were impaired only when the tone signalled negative trials, but not when it signalled positive trials.

Very recently, Plunkett and Faulds (1979) have shown that hippocampal lesioned rats are severely impaired on a successive go, no-go task in a straight-alley runway when required to discriminate between horizontal and vertical stripes or between black and white stimuli presented in the goalbox, but are not impaired on a tactile discrimination in which the stimuli, rough or smooth sandpaper, or a hot or cold metal surface, are presented in the runway.

Thus, although some of the procedural differences appear to be important, in many cases, cats and rats with hippocampal lesions are impaired on the acquisition of a successive go, no-go discrimination task by their reduced ability to withhold responses to the negative stimulus. In one experiment (Swanson and Isaacson, 1967), a similar deficit was also found in rats on reversal.

b) Responding on various schedules of reinforcement

Since response inhibition or timing behaviour appeared to be affected by hippocampal lesions, a number of experiments were carried out to study the effects of lesions on various schedules of reinforcement. Ellen and Powell (1962) trained unoperated cats and rats with small anterior hippocampal lesions on a continuous reinforcement (CRF) schedule followed by an FI 1 min schedule of reinforcement. Overall, there was no difference in the response rates of the two groups, but in the 10 seconds just prior to reinforcement the hippocampal rats tended to respond at a lower rate than the controls. However, immediately following a reinforcement the hippocampal animals were impaired in their ability to withhold a response, their mean post-reinforcement pause (PRP) being significantly shorter than that of the control rats. Haddad and Rabe (1969) trained two groups of hippocampal rats, one

with small anterior lesions and the other with larger anterior and posterior lesions, and a group of sham-operated rats on CRF and then on an FI 1 min schedule. Compared with the other two groups, the rats with the larger lesions responded at a significantly higher rate overall and in the period just prior to reinforcement. There were, however, no differences between the three groups immediately following a reinforcement, although from their graphs it appears that the rats with large hippocampal lesions, and to a lesser extent the anterior hippocampal rats, tended to make more responses in this period than the sham-operated rats. In contrast, the rats with the smaller, anterior lesions did not differ from the controls on any of the measures used, although they did show a tendency to respond at a lower rate than the controls immediately prior to reinforcement. Earlier, Rabe and Haddad (1968) had investigated the effects of either anterior or combined anterior and posterior hippocampal lesions on performance on an FR 20 schedule following CRF pretraining. They found that both hippocampal groups responded at a significantly higher rate than the sham-operated controls on the FR 20 schedule, but during CRF training only the rats with the large hippocampal lesions responded significantly faster than the controls. Also, only this hippocampal group showed a significant increase in response rate on transferring from CRF to FR 20. In two experiments carried out by Jarrard (1965), it was found that in either the postoperative retention or the acquisition of stable responding on a variable interval (VI) 2 mins schedule of reinforcement, hippocampal rats responded at a significantly higher rate than cortical or unoperated controls. More recently, Schmaltz et al (1973) trained rats preoperatively on CRF followed by one day on each of FR 5, FR 10, FR 20, FR 40, and FR 80, and finally five days on FR 160. Some of the rats were then given bilateral hippocampal lesions, while others were sham-operated or unoperated controls, and they were all retrained on the same sequence of schedules as before. When the pre-and post-operative scores were compared it was found that the

hippocampal rats showed a greater postoperative increase in response rate on each of the FR schedules compared with the control groups, but that these differences were significant only for the FR 80 and the first three FR 160 sessions.

Once again, therefore, evidence was obtained which showed that hippocampal lesioned rats suffered from some sort of response disinhibition, since they responded more, or faster, than normal rats on a simple operant task.

c) Simultaneous discrimination tasks

Hippocampal deficits on the acquisition and reversal of successive discrimination problems and inappropriate, perseverative, responding in other tasks have led a number of investigators to study the effects of hippocampal lesions on a variety of simultaneous discrimination tasks. Thompson and Langer (1963), using footshock in a T-maze, trained rats preoperatively on the acquisition of a position discrimination, followed by eight reversals. Postoperatively, the rats were each given twenty position reversals and, compared with the unoperated and neocortical controls, the hippocampal rats were slower to learn each reversal, due largely to perseverative responding to the previously correct position. It should, perhaps, be pointed out here that several other groups of rats, including those with septal lesions, preoptic hypothalamic lesions, and lesions of the medial cortex anterior to the corpus callosum, were also similarly impaired. In a related task, Kimble and Kimble (1965) trained hippocampal rats, and neocortical and unoperated controls on the acquisition and reversal of a position discrimination, using food reward in a Y maze. Initially, five trials were given to determine position preferences, and the nonpreferred position was correct in acquisition. All three groups learned the original position habit in approximately the same number of trials, but the hippocampal rats were severely impaired on reversal, taking significantly more trials to reach criterion on the first reversal, and achieving fewer reversals in 100 trials, than either of the two control groups. As before, the hippocampal deficit was due to

their perseverating the initially learned position response much longer than the controls. It has also been found that monkeys with hippocampal lesions (Mahut, 1971) or with transection of the fornix-fimbrial system, one of the main afferent and efferent pathways of the hippocampus (Mahut, 1972), are impaired on the reversal, but not the acquisition, of a position discrimination when trained in a Wisconsin apparatus. However, Samuels (1972) carried out an experiment which was similar to that of Kimble (1965) and found that her hippocampal rats were impaired on both the acquisition and the reversal of a position discrimination.

Kimble (1963) trained hippocampal lesioned rats on the acquisition of a simultaneous brightness discrimination in a Y-maze and found that they took no more trials to reach criterion than either the neocortical or the unoperated control rats. Similarly, Silveira and Kimble (1968) found that hippocampal rats were unimpaired on acquisition , but were severely impaired on the reversal of a simultaneous brightness discrimination, taking significantly more trials to criterion on the first reversal compared to the neocortical or unoperated controls, and making significantly fewer reversals in 100 trials. Although the hippocampal animals made significantly more perseverative responses to the previously correct stimulus at the beginning of the reversal than the other two groups, their reversal deficit was mainly due to the very large number of position responses that they made. Samuels (1972) also trained hippocampal rats on the acquisition of a brightness discrimination in a Y-maze, with the nonpreferred (white) stimulus being positive, and then on three reversals. In this experiment none of the hippocampal rats was impaired on the acquisition of the task, and the rats with small to moderate combined anterior and posterior hippocampal lesions were also unimpaired on the three reversals, but the rats with large hippocampal lesions made significantly more errors over the three reversals, and also tended to make more errors on acquisition.

The effects of hippocampal lesions on pattern discriminations have been studied by Douglas and Pribram (1966) using rhesus monkeys. They were trained postoperatively on an operant task in which various stimuli could be projected on to press-panels, and they learned to discriminate between large and small yellow circles as readily as the sham-operated controls. However, on reversal, the hippocampal monkeys took more trials to reach criterion, and their scores were consistently below those of the controls. Schram (1971) has also looked at the effects of hippocampal lesions on pattern discrimination and reversal learning, but in squirrel monkeys. She used compound stimuli containing three components (background orientation, form, and colour), and found, unexpectedly, that monkeys with hippocampal or fornix lesions learned the discrimination and the reversal consistently faster than the sham-operated controls. Previously, Mahut had found that hippocampal lesioned monkeys (1971) and monkeys with transection of the fornix-fimbrial system (1972), when trained in a Wisconsin apparatus, were not impaired on the acquisition or the reversal of an object discrimination, in which the stimuli were a red plastic tomato and three large paper clips.

Teitelbaum (1964) and Webster and Voneida (1964) have both investigated the effects of hippocampal lesions in cats on their performance on the acquisition and reversal of a tactile discrimination. The same apparatus was used in each case, and in it the cats were required to choose and press one of two pedals on the basis of the tactile cues that were present. In both experiments it was found that the hippocampal cats learned the initial discrimination as well as normal and cortical lesioned controls, but that they were markedly impaired on reversal. However, an interesting finding from Teitelbaum's experiment was that cats with orbitofrontal lesions, although unimpaired on acquisition, showed the same type of deficit on reversal as the hippocampal cats.

It is clear, therefore, that as a rule, hippocampal lesioned rats, cats, and

monkeys are not impaired on the acquisition of a variety of simultaneous discrimination tasks, presented in several different types of apparatus, but that they are impaired on reversal learning. However, there are two important points to note here. First, it is also clear that there are exceptions in each case, although the finding of a deficit on acquisition appears to be much less common than the finding of a lack of impairment on reversal. Secondly, a reversal learning deficit is not uniquely the result of hippocampal lesions, but can also occur following discrete lesions in other areas of the brain.

d) Extinction

The effects of hippocampal lesions on extinction behaviour have been investigated in a variety of situations. Isaacson et al (1961) compared a group of hippocampal rats with cortical and unoperated control groups on the extinction of an active avoidance response in a shuttle box, and found that the hippocampal rats had significantly shorter response latencies to the CS (a buzzer) than either control group. Jarrard et al (1964) trained rats postoperatively on a simple food-reinforced response in a runway under conditions of either massed or distributed practice, and then on the extinction of the response. Unexpectedly, the cortical controls ran significantly faster than either the unoperated controls or the hippocampal rats in acquisition, but during extinction the hippocampal rats generally ran faster than the control animals. However, only the hippocampal rats given distributed practice were significantly impaired in extinction. Niki (1965) gave rats three days of operant training on a CRF schedule followed by three days of extinction trials. Postoperatively they were given a further two days of extinction trials, and it was then found that hippocampal rats responded at a higher rate and for longer than cortical controls. Henke and Bunnell (1971) also used a simple operant response, in which hippocampal, amygdaloid, and cortical, sham-operated, and unoperated control rats were reinforced on a CRF schedule, after which they were given extinction trials for five days. Throughout the extinction sessions the hippocampal rats made significantly more responses than any of the other groups.

The extinction deficit following hippocampal lesions has been shown to occur in cats by Peretz (1965), who trained a neocortical control group and a hippocampal group to open a small window to obtain food, and then attempted to extinguish their responses. Although the two groups did not differ in their response latencies during training, the hippocampal cats had very much shorter latencies during extinction. Also, Douglas and Pribram (1966) found that hippocampal monkeys, after overtraining on a 70:30 visual probability task, were severely impaired in extinction, their response latencies being consistently shorter than those of either the sham-operated or the amygdaloid lesioned groups. Nevertheless, there have also been a number of reports in which rats with hippocampal lesions did not show an increased resistance to extinction (e.g., operant tasks: Schmaltz and Isaacson, 1967, and Nonneman et al, 1974; conditioned suppression task: Nadel, 1968; active-avoidance task: Ackil et al, 1969).

It can therefore be seen that, following training on a variety of tasks, and in different apparatus, hippocampal lesions in rats, cats, and monkeys can give rise to a marked extinction deficit in which the animals continue to respond faster, and for longer periods, than normal animals or animals with certain other types of lesion. However, it is also clear that hippocampal animals are not necessarily impaired in extinction tasks.

e) Passive avoidance tasks

One of the earliest reports of a hippocampal deficit on an avoidance task was that of Kimura (1958), who found that small posterior, but not anterior, hippocampal lesions in rats impaired the acquisition and retention of a one-trial passive avoidance response. Since then numerous reports have confirmed the finding of a passive avoidance deficit in hippocampal rats, although there have also been a number of contradictory reports. Isaacson and Wickelgren (1962) gave hippocampal or neocortical lesioned rats 60 trials in an apparatus in which they had to run from a large compartment into a small compart-

ment in order to obtain food, but on trial 35 the rats were shocked while eating. Subsequently, on trials 36-60, the neocortical controls were very reluctant to enter the small compartment to feed, their response latencies now being very much longer than before, whereas the effect of the shock on the hippocampal animals was only transient, and their response latencies were no longer than they were before shock was administered. Kimble (1963) trained an unoperated and two lesioned groups of rats for 23 trials to obtain food in the goal box of a straight-alley runway, and on the 24th trial the rats were given shock while eating. Although there had been no differences in the preshock latencies between the three groups, the mean postshock latencies of the hippocampal rats during trials 25-40 were significantly shorter than those of the normal rats. However, the postshock latencies of the cortical lesioned rats were also shorter than those of the normal rats, but were not significantly different from those of either the normal or the hippocampal rats. Later, Kimble et al (1966) trained hippocampal rats and cortical lesioned rats on a similar runway task for water reward before being given avoidance training, and again found that the hippocampal rats were impaired. But they also tested two further groups of rats on a spontaneous 'step-through' passive avoidance task in which they had not been given prior approach training, and found that there were no significant differences between the step-through latencies of the hippocampal and cortical control groups on any of the four days of testing. They therefore suggested that the initial approach training was an important factor in the occurrence of a hippocampal passive avoidance deficit, and this was confirmed in an experiment by Stein and Kirkby (1967), who found that hippocampal rats given 5 days of approach training before receiving shock were not impaired in their postshock avoidance responding, but that hippocampal rats given 10 days of preshock training were subsequently impaired. However, Isaacson et al (1966) found that, even without prior training, hippocampal rats were impaired in a runway

passive avoidance task compared with cortical and sham-operated controls, but that hippocampal rats receiving 20 or 40 training trials were more impaired.

Recently, Winocur and Black (1978) have confirmed that hippocampal lesioned rats show a marked passive avoidance deficit in a straight runway in which initial exploratory behaviour and 45 trials of water-rewarded approach training were followed by 5 trials in which shock was administered while the rats were drinking in the goal box. A similar deficit was also found to occur in a recall test given 24 hours later. However, if the rats were exposed to shock and related cues, or to shock-related cues only, 2 hours before the recall test, the passive avoidance deficit did not occur, and the performance of the hippocampal rats was no different from that of the cortical and sham-operated controls.

Finally, there is evidence that passive avoidance behaviour is affected by hippocampal lesions in mammals other than rats. Papsdorf and Woodruff (1970) found that hippocampal lesioned rabbits, when trained on a passive avoidance task in a shuttle box in which they had previously received two-way active avoidance training, took significantly more trials to reach criterion than either the cortical lesioned or the unoperated controls. More recently, Nonneman and Isaacson (1973) have shown that cats with hippocampal lesions are also impaired on a passive avoidance task.

Thus, it can be seen that passive avoidance deficits occur in hippocampal lesioned rats, rabbits, and cats in a variety of testing situations, particularly if the animals are given previous approach training in the apparatus. However, as Isaacson et al (1966) have shown, prior training is not a necessary prerequisite for a deficit to occur, and conversely, Winocur and Black (1978) have shown that, even with a reasonable number of approach trials, under certain conditions a passive avoidance deficit in hippocampal animals need not occur.

In this brief review it has been shown that those problems on which birds with

combined hyperstriatal and hippocampal lesions are impaired are precisely those on which at least several species of mammals with hippocampal lesions can be impaired, including, variously, rats, rabbits, cats, and monkeys. Furthermore, the generality of the deficits can also be seen in the fact that they occur in a variety of experimental situations, as well as in the different species. However, a number of experiments have been cited in which a hippocampal deficit was not found on a particular task, despite contrary findings by other investigators. Although procedural differences or task variables are obviously important, in many cases it is not easy, or indeed possible, to specify with any certainty the factors which are most likely to have contributed to the different results. It should, perhaps, be pointed out that this state of affairs is not unique to the study of hippocampal function in mammals, and that many examples of contradictory findings may be found in the field of physiological psychology (for example, see Grossman, 1973 passim). A further point which it is important to note, and which has already been made elsewhere in this chapter, is the observation that some of the deficits that result from hippocampal lesions can also be found following lesions in other parts of the brain.

Having reviewed the various studies of the hyperstriatal complex and the relevant behavioural studies of the mammalian hippocampus, it is appropriate now to discuss the detailed structure of the hippocampal formation in mammals, birds, and reptiles in order to be able to determine the extent to which the avian and the mammalian hippocampus can be regarded as homologous structures.

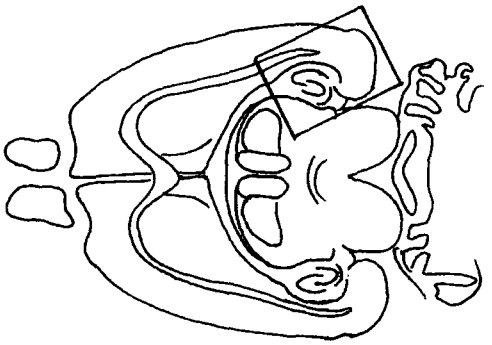
The anatomy of the mammalian hippocampus

The hippocampal formation of the mammalian brain is composed of phylogenetically older cortex, or allocortex, and is usually understood to consist of the hippocampus proper (cornu ammonis), the dentate gyrus (or fascia dentata), and the subiculum (Isaacson, 1974; Angevine, 1975), although the parahippocampal gyrus is sometimes

included in place of the subiculum (MacLean, 1975; Powell and Hines, 1975), and conventionally it is the hippocampal formation that is being referred to whenever the term hippocampus is used. In higher mammals such as the rat, the hippocampus is a very prominent curved structure which forms a semicircle in the vertical plane around the thalamus. That part of the hippocampus which lies over the thalamus has been referred to by many authors as dorsal hippocampus, and the more ventral portion, which extends into the inferior horn of the lateral ventricle, has been termed ventral hippocampus, and there is, in fact, evidence from anatomical, biochemical, and behavioural studies which clearly indicates that there are functional differences between these two regions of the hippocampus, (for example, see Livesey, 1975).

A diagram of a horizontal section through the vertical portion of the semicircular hippocampus is presented in Figure 5. From this it can be seen that it derives its very characteristic appearance primarily from two curved and interlocking bands of cells, the stratum pyramidale of the hippocampus proper and the stratum granulosum of the dentate gyrus. The dentate gyrus contains granule cells densely packed in several layers within the stratum granulosum, with their apical dendrites extending outwards towards the hippocampal fissure. Inside the V-shaped formation of the dentate gyrus, next to the granule cell layer, are several layers of polymorphic cells, the first layer of which makes up the area known as the hilus of the fascia dentata. Also found in this area are a large number of pyramidal cells, and at present it is unclear whether this region is strictly part of the dentate gyrus or the hippocampus proper, although it clearly constitutes a transition zone between the two. Extending out from this area and forming a horseshoe shape is the pyramidal cell layer of the hippocampus proper, which curves round past the fimbria and the overlying alveus to form a border with the subiculum.

On the basis of cell type and structure, the anatomist Lorente de Nó (1934)



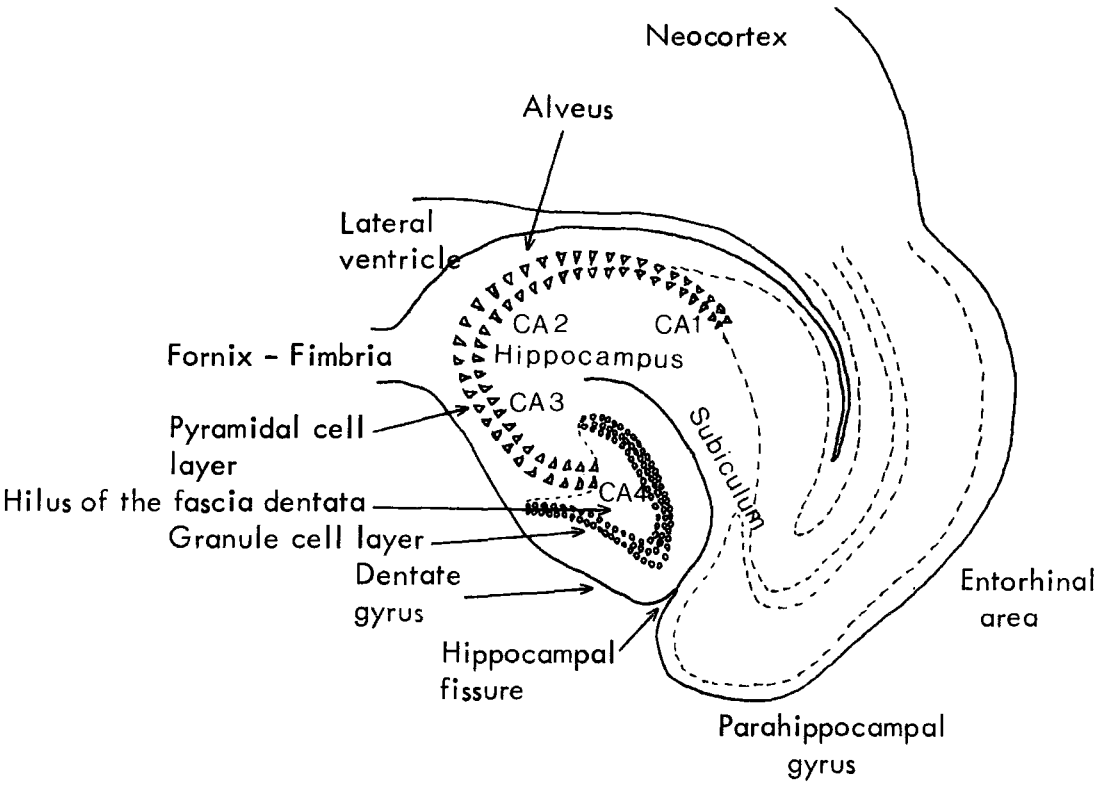


Figure 5. Diagram of a horizontal section through the right hippocampal formation of the rat (adapted from Isaacson, 1974). The frontal pole of the brain lies towards the top left-hand corner of the page - see inset diagram of a horizontal section through the brain of the rat (redrawn from Zeman and Innes, 1963).

further subdivided the hippocampus (cornu ammonis) into four fields, labelled CA1 to CA4. Field CA1 is the region which borders on to the subiculum and contains small pyramidal cells densely packed in a single layer. Next to this is CA2, which Lorente de Nó (1934) regarded as a transition zone between CA1 and CA3, and according to Isaacson (1974), some authors have considered it not to be a distinct region of the hippocampus, although Angevine (1970) has shown that, during embryological development, the cells in CA2 are formed at a different time from those in CA1 and CA3. Field CA3 is made up of large pyramidal cells, and finally CA4 is the small region of pyramidal cells in the hilus of the fascia dentata.

Unlike the pyramidal cells of the neocortex, which each have a single apical dendrite that extends towards the surface of the brain, the hippocampal pyramidal cells have both apical and basal dendritic arborizations which extend out from the cell towards the outer and the inner regions of the hippocampus proper, and are often referred to as double pyramidal cells. The axons of these pyramidal cells descend into the alveus where some continue along and into the fornix, while others bifurcate, one branch leaving via the fornix and the other continuing in the alveus for some distance or in the layer immediately next to the stratum pyramidale for a short distance before terminating. It has been shown by Raisman et al (1966) that, in the rat, the hippocampal fields of Lorente de Nó, besides differing cytoarchitectonically, also show differences in their fibre projections and terminations, although Isaacson (1974) has pointed out that Siegel and Tassoni (1971) were not able to find such regional differences in fibre distribution in the hippocampus of the cat. Next to field CA1 is the subiculum, which is not laminated but is composed of medium-sized and large pyramidal cells dispersed over a fairly wide area.

The area labelled parahippocampal gyrus in Figure 5 consists of three separate regions which, from the beginning of the hippocampal fissure round towards the

entorhinal area, are the presubiculum, the retrosplenial area, and the parasubiculum, which merges imperceptibly into the entorhinal area. The beginning of the transitional zone between the neocortex of the cerebral hemispheres and the allocortex of the hippocampal formation is marked by the entorhinal area which, together with the parahippocampal gyrus, comprises the juxtallocortex.

The main fibre system of the hippocampus is the fornix-fimbrial system which, in addition to the commissural fibres which connect the hippocampal regions in the two hemispheres, contain afferent and efferent fibres that project between the hippocampus and the septal area, the preoptic and hypothalamic areas, and other areas via the medial forebrain bundle. Important projections to the hippocampus also come from the entorhinal area by way of the perforant path and the alveus pathway.

The anatomy of the avian hippocampus

It would appear that the presence of hippocampus in the avian brain was first recognized by Rose in 1914, when he described a narrow band of cells lying in the dorsomedial wall of the hemisphere, which he referred to as the cornu ammonis. Dorsal to this, in the upper medial wall of the hemisphere, he also observed four-layered cortex composed of pyramidal cells, which he called the entorhinal area (or 'Hippocampusrinde') on the basis of its similarity in appearance to the entorhinal area in the mammalian brain.

In their extensive study of the nuclei and fibre tracts in the avian brain, Huber and Crosby (1929) described, in the brain of the English sparrow (Passer domesticus), a band of granule cells occupying the narrow region between the ventricle and the medial wall of the hemisphere and extending anteriorly from just in front of the anterior pole of the ventricle, in the region of the olfactory bulbs, to the level of the taenial nucleus in the posterior part of the hemisphere. They referred variously to this band of cells as hippocampus, hippocampal area, or hippocampal formation, and

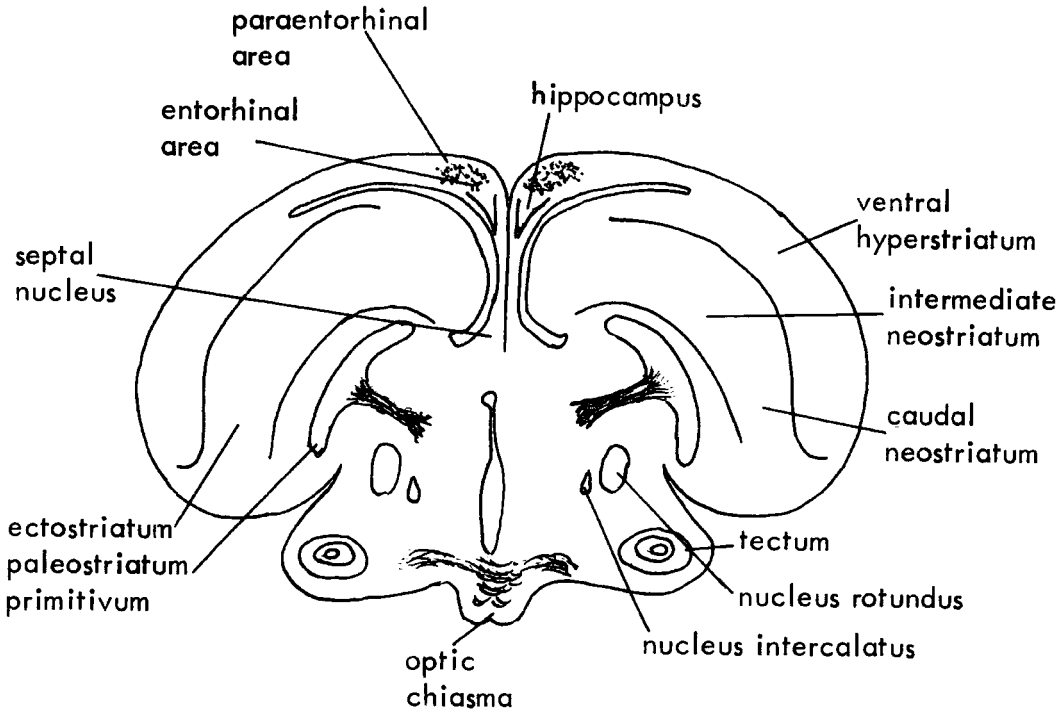


Figure 6. Coronal section of the brain of the sparrow (redrawn from Huber and Crosby, 1929).

clearly regarded it to be the same as the cornu ammonis of Rose (Figure 6; see also Figure 2).

Lying above this, in the more dorsal region of the medial wall, they were able to distinguish a scattered group of cells in the anterior region of the hemisphere, which then rapidly increased in number, becoming definitely pyramidal in character. The arrangement of these cells was found to form a long ovoid shape which, in the sparrow, was not always clearly distinguishable from the underlying hippocampal area. In the anterior region of the hemisphere this structure is situated between the hippocampal area and the overlying accessory hyperstriatum, whereas posteriorly it extends up to the dorsal surface. They referred to this group of pyramidal cells as the entorhinal area of Rose, although, as they were careful to point out later (Ariëns Kappers et al, 1936), they had done so reluctantly because they had been unwilling "to add to the already overburdened nomenclature" (p.1373), but at the same time had not wanted to imply any homology with the mammalian entorhinal area. Huber and Crosby (1929) did, however, compare it with the similarly placed hippocampus pars dorsalis in the reptile brain and later (Ariëns Kappers et al, 1936) confirmed their preference for the use of this term in place of entorhinal area (of Rose).

Dorsolateral to the hippocampus pars dorsalis Huber and Crosby (1929) identified a layer of pyramidal cells which they called the paraentorhinal area. In the anterior region, it is situated between the hippocampus pars dorsalis and the accessory hyperstriatum in the angle between the medial wall of the hemisphere and the ventricle. Posteriorly it widens laterally and subsequently, with the disappearance of the accessory hyperstriatum, becomes continuous with the area they label as dorsolateral surface area (corticoid) in their figures but which they describe as "cortex-like lamina of the dorsal wall" (p. 60). Essentially the same account of these dorsomedial and dorsolateral regions of the avian brain was provided by Ariëns Kappers et al (1936), although the

paraentorhinal area was renamed area X "in the lack of any precise knowledge of its mammalian homologies" (p. 1378). Area X has since become known as the parahippocampal area, and the dorsolateral surface area (corticoid) is now called the dorsolateral corticoid area.

The main fibre tract from the hippocampal area, the septomesencephalic (or corticoseptomesencephalic) tract (TSM) has been described by Ariëns Kappers et al (1936) as arising partly from the accessory hyperstriatal and associated regions in the rostral part of the forebrain (i.e., predominantly the Wulst) and coursing caudally and ventrally towards the medial wall where it is joined by fibres from the parahippocampal and hippocampal areas. It then passes through the septal area, where it is joined by other fibres. Subsequently a bundle called the ramus basalis frontalis is given off, which is homologous with the pathway in reptiles that projects between the hippocampus and the amygdala. A further major division is the ramus basalis caudalis, which terminates in the hypothalamic area and is part of the fornix longus. More recent details of the anatomy of the avian fornix-fimbrial system are discussed on p. 73.

Comparison with the hippocampal formation of reptiles

The hippocampal cortex in many reptiles is divisible into a dorsomedial portion, the hippocampus pars dorsomedialis, and a dorsal portion, the hippocampus pars dorsalis (see Figure 7). The hippocampus pars dorsomedialis, also referred to as fascia dentata in the turtle (Cistudo carolina) by Johnston (1915), consists of a closely packed layer of cells lying between the ventricle and the medial wall of the hemisphere. In the lizard these are granule cells, but in turtles and the crocodilians they are pyramidal cells. In the alligator (Alligator mississippiensis), Crosby (1917) described large double pyramidal cells in this region. Nevertheless, Ariëns Kappers et al (1936) state that the dorsomedial portion as described in the lizard (Lacerta agilis)

by Ariëns Kappers (1909) appears to be homologous with the fascia dentata in the turtle and the hippocampus dorsomedialis in the alligator. Huber and Crosby (1929) and Ariëns Kappers et al (1936), on the basis of their own findings and of the earlier evidence of others (e.g., Elliot Smith, 1910; Herrick, 1910; Crosby, 1917), state that this part of the hippocampal formation is the homologue of the cornu ammonis of Rose, or the hippocampus of Huber and Crosby, in the avian brain, and the phylogenetic origin of the mammalian fascia dentata, (a proposal that had been made earlier by Johnston, 1915) . To facilitate comparison, the various names employed by different authors for corresponding areas of the hippocampal formation in reptiles, birds, and mammals, and the proposed homologies between them, are presented in Table 1.

The hippocampus pars dorsalis, which in the turtle Johnston called the subiculum cornu ammonis, lies in the medial wall of the hemisphere next to the ventricle dorsal to the hippocampus pars dorsomedialis, and consists of a loosely packed group of pyramidal cells. Ariëns Kappers et al (1936) regarded the subiculum in the turtle and the hippocampus pars dorsalis in the alligator to be homologous, but were not exactly sure of the relationship of either of these two areas to the dorsal hippocampus in the lizard. This region of the hippocampal formation in reptiles was regarded by Huber and Crosby (1929) and Ariëns Kappers et al (1936) as the reptilian homologue of the avian hippocampus pars dorsalis, or the entorhinal area of Rose, and also to be the origin of the cornu ammonis or hippocampus proper in mammals (see Table 1).

Dorsolateral to the hippocampus pars dorsalis is a further layer of cells, which Ariëns Kappers et al (1936) refer to as differentiated cortex, and which Dart (1920) had earlier called parahippocampal cortex. It extends from the extreme dorsal region of the medial wall of the hemisphere over on to the dorsal area and becomes continuous with the general cortex of the dorsolateral surface.

Several bundles of fibres extend down from the dorsal and dorsomedial regions of

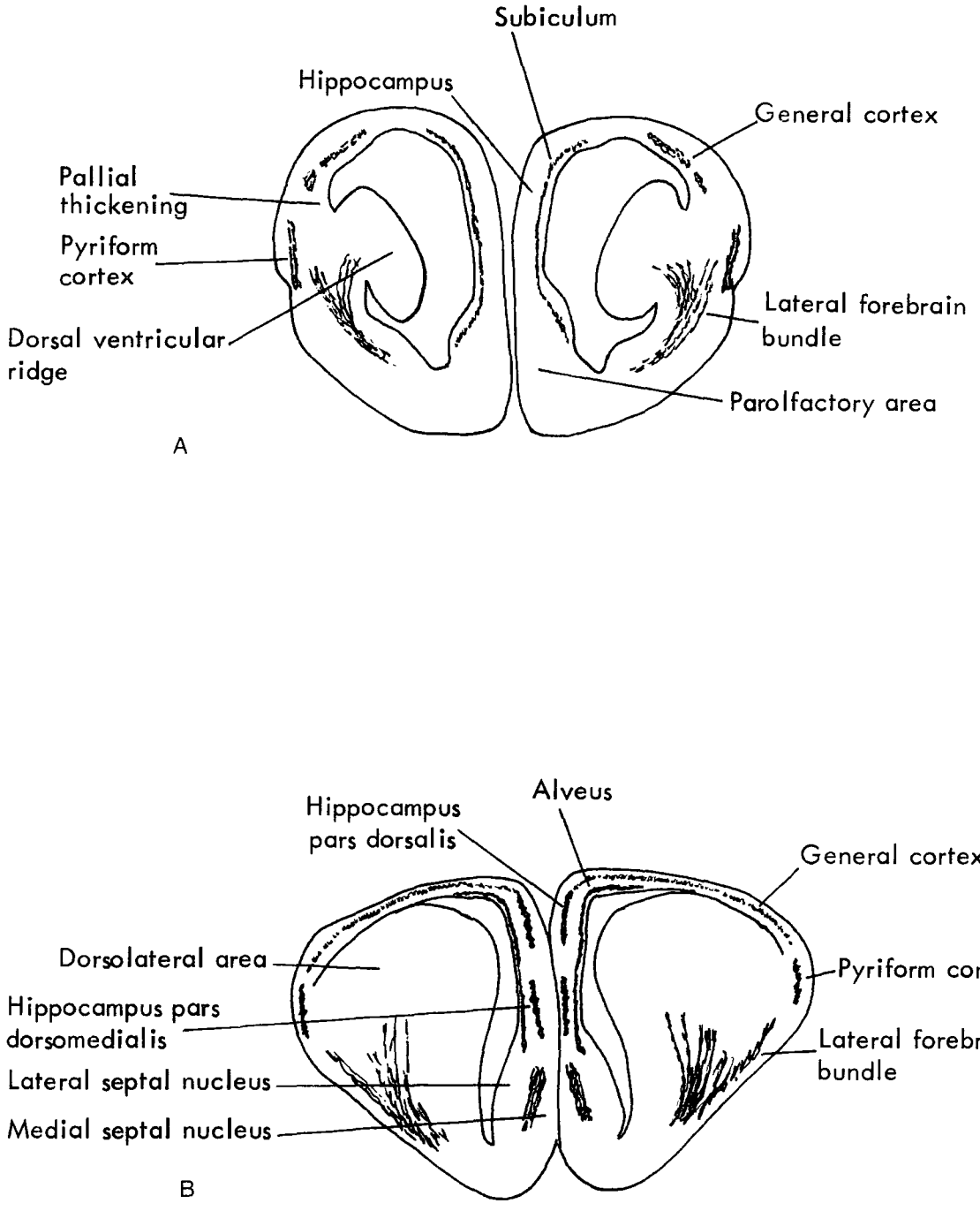


Figure 7. Coronal sections through the brains of A. the turtle (Pseudemys scripta) (Redrawn from Hall and Ebner, 1970), and B. the alligator (Alligator mississippiensis) (Redrawn from Ariëns Kappers et al, 1936).

Table 1

Table of proposed homologies between hippocampal areas in reptiles, birds, and mammals.

Mammals	Reptiles	Birds
Fascia dentata Granule cells	Dorsomedial hippocampus (Ariëns Kappers, 1909; lizard) Granule cells	Cornu ammonis (Rose, 1914; various species) Granule cells
	Fascia dentata (Johnston, 1915; turtle) Pyramidal cells	Hippocampus (Huber and Crosby, 1929; sparrow) Granule cells
	Hippocampus pars dorsomedialis (Crosby, 1917; alligator) Double pyramidal cells	Fascia dentata (Craigie, 1930, 1932; kiwi, humming bird) Granule cells
Cornu ammonis Double pyramidal cells	Dorsal hippocampus (Ariëns Kappers, 1909; lizard) Pyramidal cells	Entorhinal area (Rose, 1914; various species) Pyramidal cells
	Subiculum cornu ammonis (Johnston, 1915; turtle) Pyramidal cells	Hippocampus pars dorsalis (Huber and Crosby, 1929; sparrow) Pyramidal cells
	Hippocampus pars dorsalis (Crosby, 1917; alligator) Pyramidal cells	Subiculum (Craigie, 1930, 1932; kiwi, humming bird) Pyramidal cells

the hemisphere in reptiles, via the hippocampal and septal areas, and project to the hypothalamus and other diencephalic and mesencephalic areas. One of the fibre tracts is the diagonal band of Broca, which forms a connection between the hippocampal area and the amygdaloid complex in reptiles, and is homologous with the avian ramus basalis frontalis. The other fibre systems include the cortico-hypothalamic tract and the fornix longus, which together appear to be representative of the fornix system in mammals and of the TSM in birds.

The question of homology

The earlier comparative anatomists used a variety of techniques in their attempts to establish homologies between various structures in the brains of mammals, reptiles, and birds. These included detailed studies of gross morphology, the topographical relationships of cell groups and fibre tracts, and the microscopic examination of cell types and their distribution and fibre connections, together with evidence from studies of the migrations of cell groups or layers during embryological development. It is important to note, however, that these earlier studies all relied on normal histological material.

In recent years the development of more advanced techniques in electrophysiology, degeneration methods, autoradiography, histochemistry, and other highly sophisticated biochemical approaches have made it possible to identify the characteristics of particular cell types or groups and to trace and analyse fibre connections with far greater precision and in much greater detail. Using these techniques, a number of important new findings have been made by, for example, Hodos and Karten and their colleagues concerning the organisation and projection of fibres to and from the various cell groups in the avian brain that comprise the so-called corpus striatum. Some of this work was discussed earlier in this chapter. These findings have not only considerably increased our knowledge of the 'unusual' organisation of the avian brain, but have

also advanced our understanding of its relationship to the organisation of the more familiar mammalian brain. However, while providing confirmation of many of the earlier anatomical observations, it is also the case that a number of these recent studies have given rise to interpretations that differ radically from those of the earlier workers. For example, it had, in the past, been proposed that the neostriatum in birds was homologous to part of the mammalian caudateputamen (part of the basal ganglia), and that the archistriatum was the homologue of the amygdaloid complex in mammals. Recent histochemical and electrophysiological studies in the pigeon have now cast considerable doubt on these proposed relationships. Karten (1969) gave reports of several studies in which it had been shown that the mammalian caudate-putamen gave a strong positive reaction for the presence of dopamine, whereas the avian neostriatum was completely devoid of dopamine; and furthermore, that a densely populated region in the medial neostriatum (Field L of Rose, 1914) has been shown (Karten, 1968) to receive a well-defined fibre tract (the tractus ovoidalis) from the nucleus ovoidalis, which is believed to be the avian homologue of the mammalian inferior colliculus. In support of this, Erulkar (1955) and others have reported auditory evoked potentials from this medial neostriatal region. On the basis of their degeneration studies, Zeier and Karten (1971) have obtained evidence to show that only a part of the archistriatum can be considered to be limbic in nature, and therefore homologous to the mammalian amygdala, and that the remainder appears to have a somatic function, and may, in fact, be comparable to the primate sensorimotor cortex.

In view of these, and other, discrepancies (see Baker-Cohen, 1968) between some of the earlier work and more recent findings it would appear that a certain degree of caution is necessary before accepting the homologies between the hippocampal formation of reptiles, birds, and mammals that were proposed by the earlier anatomists and which have been presented here. Riss, Halpern, and Scalia (1969) refer to the

countless attempts to establish homologies between various cell groups and their fibre connections in the brains of reptiles and mammals that are to be found in the literature, and clearly support the proposal, made earlier by Goldby and Gamble (1957) that "such attempts should be regarded as hypotheses in need of testing" (Riss et al, 1969, p. 1). Also of considerable importance, of course, is the question of the evolutionary relationships between reptiles, birds, and mammals (Hodos, 1970). The earliest reptiles were the Cotylosauria, from which all other reptiles evolved. Of those that survive today, the oldest are the turtles and tortoises (see Figure 8). Later, but evolving in parallel, were the Lepidosauria, the lizards and snakes. Considerably later still several other forms radiated from the Cotylosauria, one of the most important of which were the Archosauria, which gave rise to many reptile forms, including the dinosaurs. The surviving archosaurs are the crocodiles and alligators, which evolved in parallel with the turtles and lizards; but it is also believed that birds evolved from the archosaurian-stem reptiles. Fairly early on there also arose from the Cotylosauria a rather different line of reptiles. These were the Synapsida, from which evolved the now extinct mammal-like reptiles, the Therapsida, and eventually the mammals themselves. Thus, it is important to recognise that these groups of reptiles each followed independent, rather than sequential, lines of evolution, and that lizards, for example, cannot be considered to be ancestral to alligators and crocodiles. The present-day mammals are also the product of parallel lines of evolution, a point strongly emphasised by Hodos (1970); and so, too, are the birds, although it would appear that they derive from considerably fewer separate lines (Bock, 1969; Pearson, 1972).

In order to compare the brains of animals that represent divergent lines of evolution, then, it is necessary to go back to their common ancestor. Therefore, to compare, and to establish homologies between, the brains of birds and reptiles, the brain of the archosaurian-stem reptiles should, ideally, be studied and comparisons made with the

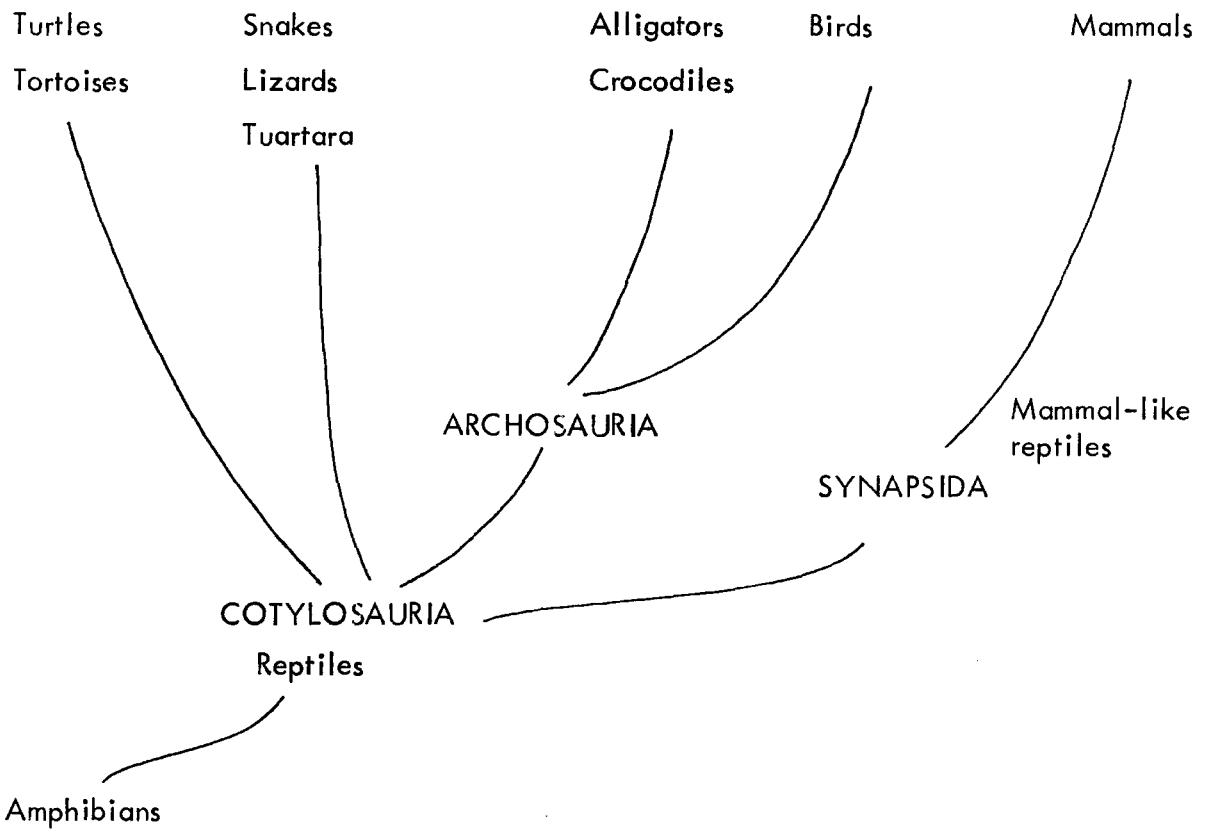


Figure 8. Diagram to show the evolutionary relationships between reptiles, birds, and mammals.

two divergent lines. Similarly, for homologies to be established between reptilian and mammalian brains, they should be compared through the primitive cotylosaurs. It follows, therefore, that homologies between avian and mammalian brains can then only properly be determined by way of the ancestral cotylosaurs. Finally, it would appear that, in order to be able to establish beyond doubt the relationships and homologies between various structures and regions in the brains of reptiles, birds, and mammals, it would also be necessary to establish homologies between the brains of the archosaurs and those of the cotylosaurs.

The ancestral-stem reptiles have long been extinct, however, and since the nervous system has never been known to provide any fossil records, the ideal strategies that are proposed above are clearly not feasible. The approach that has to be adopted instead, therefore, is to compare birds and mammals through the brains of the living reptiles that most closely resemble their common ancestors. The present-day crocodilians, having changed little since the late Triassic (some 190 million years ago), are still fairly close representatives of the reptilian ancestors of birds, and indeed, Papez (1929) has shown that the brain of the alligator is very similar in many respects to that of birds. On the other hand, turtles are the direct descendants of the most primitive stem reptiles, from which mammals are also descended. According to Riss et al (1969), however, even this approach had its fundamental difficulties, since the relationships between the brains of turtles and crocodiles had been very unclear until their work on the brain of the yellow spotted Amazon turtle (Podocnemis unifilis), a member of the more primitive of the two suborders of turtles, the Pleurodira, which apparently has changed little since the Cretaceous (approximately 140 million years ago).

By comparing the brain of this primitive side-necked turtle with the brains of members of the other suborder of turtles (Cryptodira) and the brain of caiman

(Caiman sclerops), Riss et al were able, with some confidence, to establish corresponding areas in the brains of turtles and crocodiles. Evidence that supports their work came from the studies of Baker-Cohen (1968), who found that the enzyme histochemical properties of various regions of the forebrain of caiman showed many similarities to those of both lizards and turtles. In particular, the work of Riss et al supported the earlier proposal of Ariëns Kappers et al (1936) that the regions in the turtle brain that Johnston (1915) had called fascia dentata and subiculum cornu ammonis were comparable to the regions in the brain of the alligator that Crosby (1917) had labelled, respectively, hippocampus pars dorsomedialis and hippocampus pars dorsalis (see Table 1). This was confirmed by Baker-Cohen (1969) who, using her enzyme histochemical techniques, was able to show good differentiation between dorsal and dorsomedial hippocampus in both caiman and turtle, despite the lack of cytological differentiation in the latter, as well as obtaining similar histochemical reactions in the corresponding regions of the hippocampal formation in these two reptiles. Furthermore, the fascia dentata in the turtle and the hippocampus pars dorsomedialis in the alligator were found to show histochemical activity that in general resembled that of the fascia dentata region of the hippocampal complex in the mouse, while the enzyme histochemical reactions of the subiculum cornu ammonis in the turtle and the hippocampus pars dorsalis in the alligator were very similar to those found in Ammon's horn in the mouse. According to Baker-Cohen (1969), it is believed that the mammalian hippocampal formation developed from an originally simple layer of cells by differential growth and folding, together with the separation of the cornu ammonis from the fascia dentata. Thus, although the characteristic shape of the mammalian hippocampal formation is very different from that of the hippocampal formation in reptiles, there now appears to be very good evidence from several different sources to show that they are homologous structures.

The last step, then, is to establish homologies between the avian hippocampal formation and that of reptiles and mammals, and evidence for this has been provided by the embryological studies of Källén (1962). He has shown that, by $3\frac{1}{2}$-4 days after fertilization, the cells of the embryo chick forebrain have migrated to form the dorsal telencephalic area and the ventral telencephalic area, thereby confirming his earlier observations in the pigeon embryo (Källén, 1953). He also reports that this basic division has previously been described for many vertebrates, including reptiles and mammals. During further development in mammals this dorsal area gives rise to the various subdivisions of the pallium, and from his studies of the embryogenesis of the forebrain nuclei in birds, Källén has argued that the derivatives of the avian dorsal telencephalic area should be regarded as homologous with the pallial part of the mammalian brain. Thus, according to these findings, the hippocampal formation and the parahippocampal area in birds and mammals are formed by the same cell migrations during embryological development.

Finally, the very recent autoradiographic studies of Krayniak and Siegel (1978) have demonstrated the presence of projections from the hippocampus and parahippocampal area to the septal region in the pigeon, similar to those found in the rat by Meibach and Siegel (1977). Thus, Krayniak and Siegel found projections from the caudal third of the pigeon hippocampus comparable to those that arise from CA3 in the rat; efferent fibres from the caudal parahippocampal area in the pigeon that suggest homologies with the mammalian subicular cortex, a proposal also made earlier by Benowitz and Karten (1976); and projections from the anterior third of the pigeon hippocampus to the diagonal band that are similar to the projections from the hippocampus to the diagonal band in mammals. However, two major differences were noted. First, no evidence of fibres corresponding to the postcommissural fornix in mammals was found, although Crosby and Showers (1969) described the presence of postcommissural fornix fibres in

birds, which turn downwards from hippocampal and possibly parahippocampal areas in the region of the anterior and hippocampal commissures. Secondly, Krayniak and Siegel found that the topographic distribution of the hippocampal projections to the septal area was different from that in mammals. Nevertheless, they argue that their findings strongly support the proposal that the hippocampus and the parahippocampal area in the pigeon are definitely hippocampal in nature.

Thus, there is now substantial evidence from anatomical, histochemical and embryological studies which strongly suggests that, despite millions of years of divergent evolution from the stem reptiles, the hippocampal formation in birds is homologous with that in mammals. However, Campbell and Hodos (1970) have argued that, besides various structural similarities, the establishment of homologies in the central nervous system require the demonstration of functional similarities, and this would be provided by the finding that hippocampal lesions in birds produce deficits on a variety of tasks that are comparable with those deficits that occur in hippocampal lesioned mammals.

Summary

Although the effects of lesions restricted to the hippocampal formation in birds have not yet been investigated, a limited number of experiments have been carried out to study the effects of hyperstriatal lesions, and in some of these experiments damage to the hippocampal area also occurred. A review of the various experiments revealed a number of effects that are similar to those that occur in mammals following hippocampal lesions, and a comparison of the behavioural effects of combined hyperstriatal and hippocampal lesions with the effects of lesions restricted to the hyperstriatal complex suggested that lesions of the hippocampal formation alone in birds could produce similar behavioural deficits to those that are found in hippocampal lesioned mammals.

The comparative anatomy of the hippocampal formation and its fibre connections in mammals, birds, and reptiles revealed many similarities which, together with evidence from enzyme histochemical and embryological studies on the brains of representatives of these three orders, provided strong support for the notion that the avian and mammalian hippocampal formations are homologous structures. However, in order to establish homologies in the central nervous systems of birds and mammals it is important to show that structural similarities are accompanied by functional similarities. It is proposed here that the finding that lesions restricted to the hippocampal formation in birds produce similar behavioural deficits to those that have been found to occur following hippocampal lesions in mammals would be an important contribution to the demonstration of functional similarities between the two structures.

CHAPTER 2 General Experimental Method

The experiments reported in this thesis were designed to investigate the effects of hippocampal lesions in pigeons on a variety of learning tasks using an operant conditioning paradigm. Since the details of the subjects, the apparatus used, the various preliminary procedures, and the surgical and histological techniques were identical throughout these experiments they are presented here. However, the results of the histological analysis and the reconstructions of the lesions in the individual pigeons are presented in the appropriate chapters.

Method

Subjects

The subjects were adult hybrid white pigeons (Columba livia) obtained from either an M.R.C. registered laboratory animal supplier, or a local pet shop. On arrival at the departmental animal house all pigeons, who were experimentally naive at the time, were housed individually in cages constructed of galvanised wire mesh and measuring 56.3 cms wide x 41 cms high x 46 cms deep. Two galvanised metal hoppers were fitted to the lower part of the front of the cage, one on each side of the centrally situated door, in one of which was placed food, and in the other, water. Each cage was also provided with a 13 mm diameter dowel perch, 31 cms long, fixed diagonally across one of the rear corners of the cage and 15 cms above the floor.

The animals were left relatively undisturbed for one month to allow them to acclimatise to their new surroundings. During this period they were given free access to food, a grain mixture (Fancy Pigeon Mixture B (no maples), prepared by John E. Haith and Sons, Ltd., Cleethorpes), and water, and they were weighed every other day. This was to ensure that their body-weights stabilised and that, by taking the average of the last three weighings, an accurate assessment of their free-feeding weights

was obtained. They were then selectively deprived of food, being given only 5gms of food each per day, although free access to water was maintained at all times. They were weighed daily and this restricted diet was continued until their body-weights were reduced to 80% of their free-feeding weights. Throughout each experiment all pigeons were maintained at 80% of their ad-lib body-weights by giving them in their home cages, immediately after their daily testing session, an appropriate amount of food which, together with that obtained during testing, made up their calculated daily ration.

Apparatus

The experiments were carried out using a test chamber (model CI-417A) constructed of aluminium and manufactured by Campden Instruments Ltd., London. It consisted of a pigeon panel fitted into a sound resistant housing on which was mounted an extractor fan to provide ventilation and which also served to provide a constant background masking noise (sound pressure level 75-80 dBA with respect to 2×10^{-5} Nm^{-2}, measured on a Dawe Instruments Ltd. Type 1400G Sound Level Meter). The pigeon compartment was provided with an internal clear perspex door and the whole enclosure was fitted with a drop down door containing a dark plastic one-way viewing window. The floor of the chamber was a 25mm square wire mesh grid placed over a removable droppings tray, and the internal dimensions of the animal's test compartment were 33.5cms wide x 26.0cms deep x 33.0 cms high as measured from the grid floor. The pigeon panel contained a houselight (24v, 2.8w) placed centrally near the top of the panel and, directly below it, an aperture 5.0cms wide x 6.0cms high, the lower edge of which was 10.0cms above the grid floor, which provided access to the solenoid-operated grain feeder and which was illuminated internally by a 24v 1w bulb whenever food was presented to the pigeon. The grain mixture used here was the same as that provided in the pigeons' home cages. A 6.3cm 3 ohm loudspeaker

was also mounted behind the panel by means of which auditory stimuli or white noise could be presented to the subjects.

In all of the experiments, except the first DRL 10 experiment and the visual form discrimination and reversal experiment, the panel was provided with two pecking keys (Campden Instruments Ltd., model CI-444), each 3.25 cms in diameter, with centres 15.0 cms apart and 26.0 cms above the grid floor. Each key was hinged at the upper edge and switched a reed relay when operated, the force required being 0.15 N. Behind each key was a three-colour stimulus light unit so that the keys could be independently illuminated from behind with red, green, or white light.

In the case of the two experiments referred to above, the pigeon panel was in all respects the same as that already described except that it was provided with three pecking keys. All three keys were placed in line, with their centres 26.0 cms above the grid floor. The centre key was directly below the houselight, at a distance of 5.0 cms from it, centre to centre, and the two side keys were placed on either side of the centre key, their centres being at a distance of 7.5 cms from it. In the first DRL 10 experiment the three keys were each provided with the standard three colour stimulus light unit. However, for the form discrimination experiment, each of the two side keys had mounted behind them instead a miniature inline display unit (Counting Instruments Ltd., Boreham Wood) by means of which various pattern stimuli could be back-projected on to the keys.

All stimulus and response events were presented and recorded automatically using a combination of electromechanical and solid-state programming modules, which were either manufactured by Campden Instruments Ltd., and BRD Ltd., or were built in the workshops in the Department of Psychology, The University of Newcastle upon Tyne. In some experiments the racks of control equipment were located outside the room containing the sound attenuating experimental chamber, although in the later

experiments the experimental chamber was housed in an acoustic enclosure (Amplivox Hearing Conservation Ltd.) and the programming equipment was sited nearby in the same room (see Figure 9).

Preliminary training procedure

Shortly after the experimentally naive pigeons had attained their 80% body-weights they were given several preliminary training sessions in order to adapt them to the apparatus and, except when they were due to undergo an autoshaping procedure (Brown and Jenkins, 1968) postoperatively, to train them to peck the illuminated keys and obtain reinforcement. The houselight and ventilation fan were always switched on, and on the first three days the illuminated food hopper was switched on continuously. On each day, before placing the pigeon in the apparatus, 10-15 grams of grain were put into the food hopper compartment so that it was readily visible. The pigeon was then allowed to habituate to the experimental chamber and to eat its daily ration of food there. These first three sessions usually lasted approximately 15 minutes, at the end of which the pigeon was returned to its home cage. The following day this basic procedure was repeated, except that only half the amount of food was made immediately available and the food hopper was switched off as soon as the food was eaten. Subsequently the pigeon was habituated to the noise of the food hopper when it was operated, and this session was continued either until the pigeon readily approached and ate from the food hopper whenever it was presented, or until thirty minutes had elapsed. In either case the pigeon was again returned to its home cage. This procedure was repeated on the following day for those pigeons that previously had been reluctant to respond, and food hopper training was usually successful during this session. There were, however, several pigeons which still refused to eat from the food hopper when it was operated and they were given a third training session. If this was also unsuccessful, the pigeon was returned

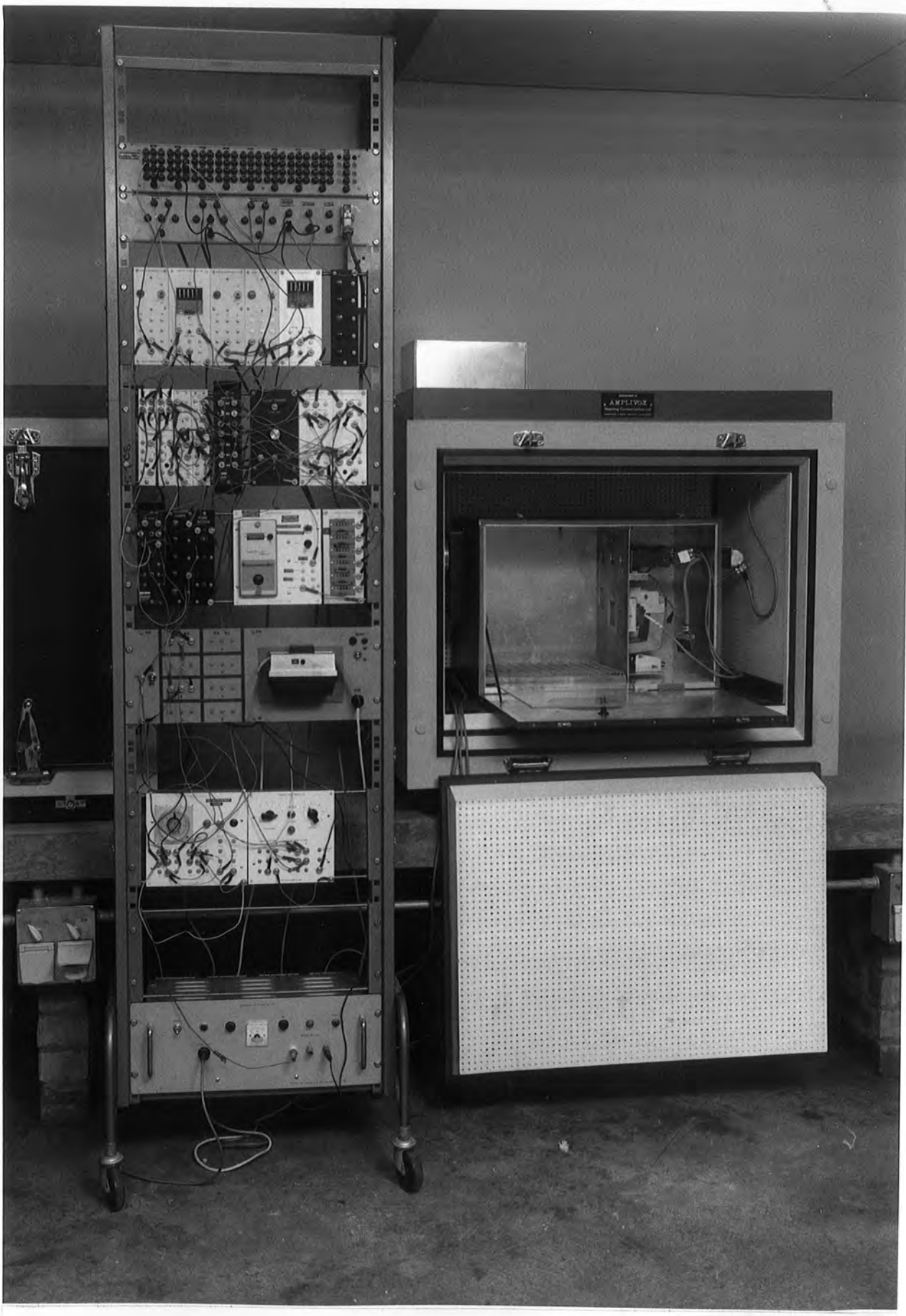
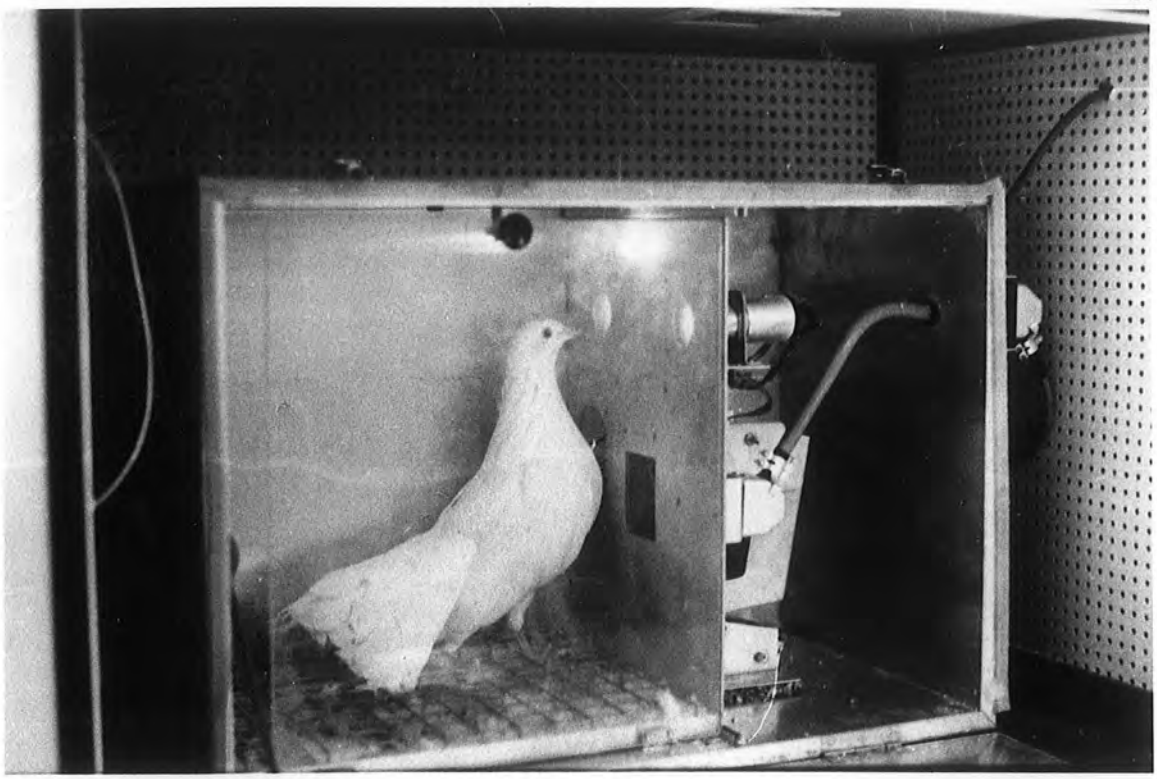
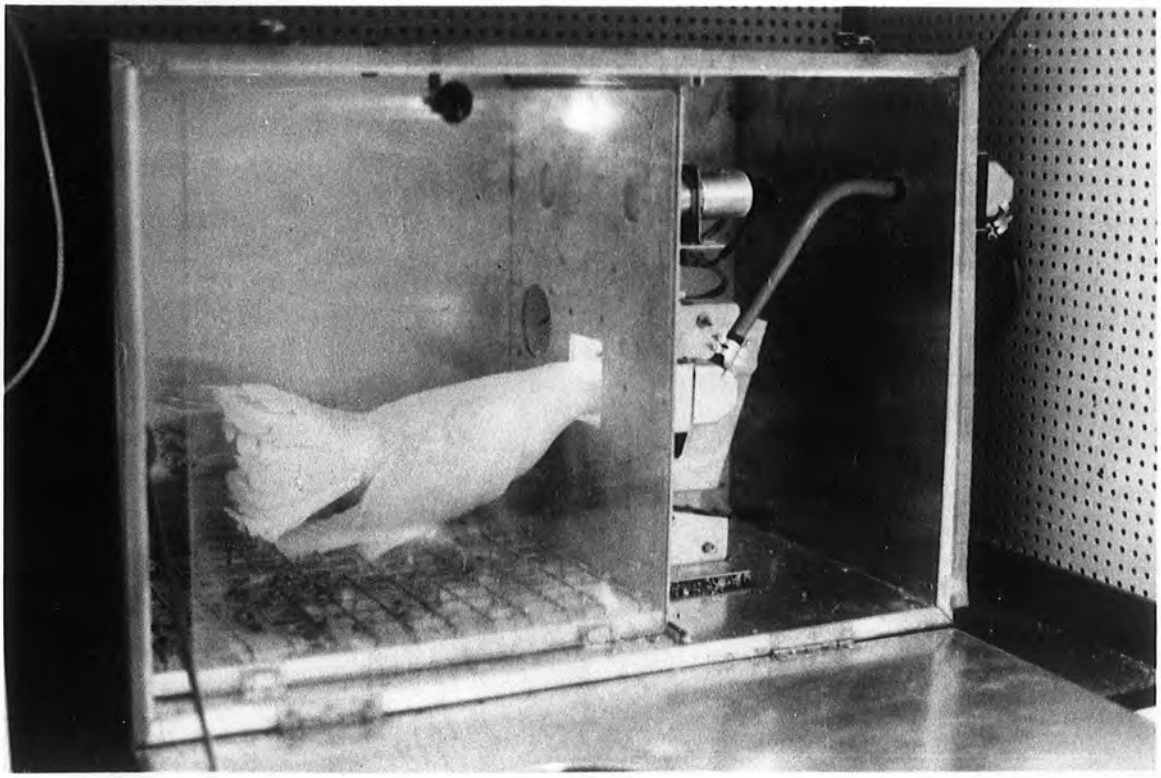


Figure 9. Photograph of a typical experimental arrangement, with the operant chamber in an acoustic enclosure and the rack of associated programming and recording equipment sited nearby.



A



B

Figure 10. Photographs of a pigeon in the operant chamber:
A. about to respond to one of the lighted keys
B. obtaining food reward following a correct response.

to its home cage, placed on a free-feeding regime, and excluded from the experiment.

As soon as the pigeons were eating readily in the experimental chamber, if they were not subsequently to be autoshaped, the second phase of preliminary training began. For this both keys were illuminated with white light and the pigeons were then handshaped by successive approximations (Ferster and Skinner, 1957) to peck at either key in order to obtain food on a continuous reinforcement (CRF) schedule (Figure 10). This session was continued for each pigeon until it had learned to peck at least one of the keys and to operate the food hopper ten times. The next day only one key was lit at a time, the other key remaining dark and inoperable, and it remained on until the pigeon pecked it and obtained food for 3 seconds. At the end of the reinforcement period one of the keys was again lit, and the order in which the two keys were presented was randomly determined. During this session each pigeon was given a total of 50 trials, each key having been presented and responded to on 25 trials. This training session was then repeated on the following day.

Surgery

The lesions were made under aseptic conditions using a Grass Instruments Co. model LM4 RF Current Lesion Maker. The indifferent electrode was a piece of smooth brass rod 15 mm long x 5mm diameter with the tip rounded and was placed in the animal's cloaca. The active electrode was a headless stainless steel insect pin 25mm long and 0.315mm diameter, insulated to within 1 mm of the tip with Schenvar 31 (Schenectady-Midland, Wolverhampton), an epoxy resin varnish. To ensure that the layer of insulation was smooth and even, the insect pin was dipped mechanically by a method used by Delius (1966). The insulating process was carried out by means of a piece of equipment, built in the departmental workshops, which basically consisted of a horizontal bar, the electrode holder, on which a number of insect pins, electrode wires, etc. could be mounted vertically and which could be

raised or lowered at a low, steady speed by means of a screwthread driven via suitable gearing by a small, reversible, variable speed, d.c. model motor (Meccano) (Figure 11). The speed of the motor was adjusted, by varying the d.c. voltage output of a Minireg power supply connected to it, so that the vertical speed of the electrode was approximately 2.5 cms per minute. Two microswitches, whose positions were adjustable, were mounted a suitable distance apart on the guidebars and were connected to the motor via a relay. The lower microswitch, which was positioned so that it was operated by the electrode holder when it reached the lowest extent of its travel, reversed the motor and the other microswitch, which was positioned at the upper extent of traverse, switched the motor off. Thus, a number of electrodes mounted on the electrode holder could be automatically dipped and withdrawn at a slow constant speed so that the coat of insulation applied would be perfectly even and free of air bubbles or droplets. Once the electrodes had been coated they were placed in a stand and baked for 15 minutes in a thermostatically controlled oven at 150°C. A total of four coats of varnish were applied, each followed by baking, the final coat being baked for approximately 45 minutes at 150°C.

The insulation was examined with the usual electrolytic bubbling technique (Silver, 1958) using 10% saline in a flat petri dish placed on the ground glass base of a low power stereomicroscope (Vickers Ltd.) and illuminated from below. The uninsulated end of the electrode was connected to the negative pole of a Minireg d.c. power supply, the output adjusted to 4.5v, and the electrode inspected for bubbling. If satisfactory, the electrode was then removed from the saline, dried, and placed on a flat disc of 5 mm thick glass on the stage of the stereomicroscope. It was now illuminated from above and, using a sharp scalpel blade, the insulation was removed up to 1 mm from the sharpened tip.

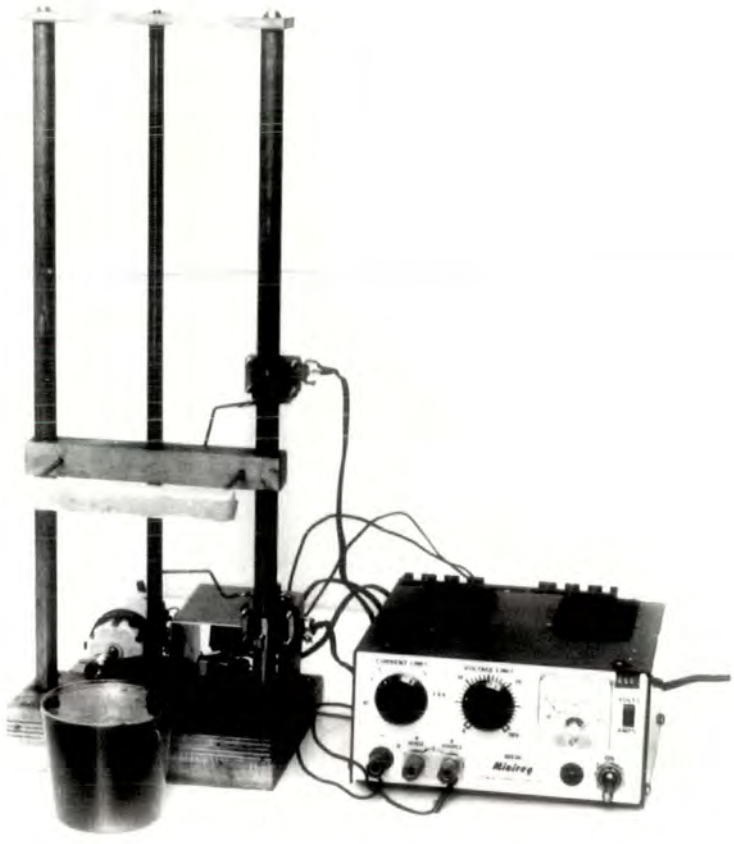


Figure 11. Photograph of the apparatus used for insulating electrodes.

The RF current lesion maker was calibrated initially using the white of a fresh egg. Both electrodes, separated by a distance of some 4-5 cms, were placed in the eggwhite. After the lesion maker had been allowed to warm up, the timer was set to 60 seconds and the intensity control set to minimum. Output from the device was then switched on and the intensity gradually increased until a volume of egg white, approximately 1mm across, surrounding the electrode tip coagulated. This was accompanied by a rapid drop in current as read from the milliammeter and a sudden rise in the voltage indicated on the voltmeter.

The stereotaxic instrument (Model 1204, David Kopf Instruments, U.S.A.) was calibrated, using a stainless steel insect pin mounted in the electrode carrier, to determine stereotaxic zero. The procedure adopted here was that described by Pellegrino and Cushman (1971).

Prior to surgery the animals were fasted for 24 hours and were anaesthetised with Equi-Thesin (Jensen-Salsbery Laboratories, U.S.A.) injected intrapectorally (0.24 mls/100 gms bodyweight). Following the anaesthetic injection, atropine sulphate (0.005 mgs/100 gms bodyweight) was injected subcutaneously to reduce mucus and saliva production thereby reducing the possibility of the animal's drowning in its own saliva. When the pigeon had reached an appropriate stage of anaesthesia, as indicated by the absence of any withdrawal reflex, it was placed in the stereotaxic instrument by first gently inserting the earbars into the external auditory meatus and then clamping them in the earbar blocks so that the pigeon's head was centrally located between the stereotaxic frame bars. To ensure greater reproducibility of head position, for both surgical and histological purposes, a Revzin adaptor (Karten and Hodos, 1967) was used in place of the standard Kopf Model 918 pigeon adaptor.

The skin on top of the animal's head was rubbed gently with a pad of cotton wool moistened with alcohol to cleanse the area of the incision and to dampen the feathers, so that they could be more easily smoothed down, and a suitable area of skin exposed. A midline incision 1.5-2 cms long was then made and the skin retracted to expose the calvarium. After scraping away the periosteum, the area of the skull to be removed was marked. From the stereotaxic atlas of Karten and Hodos (1967) the hippocampal and parahippocampal areas extend from A8.00 to A3.25, and from the midline anteriorly to L4.00 posteriorly. Using these coordinates, the electrode carrier was moved appropriately and marks made on the bone over each hemisphere to indicate the shape and extent of the required trephine hole. The bone was then drilled using a fairly blunt dental burr, the medial edge of the trephine hole being as near to the midline as possible (1.0-1.5 mm) while leaving the mid-dorsal and occipital sinuses intact, although posteriorly the trephine hole curved away from the midline, with the medial edge approximately 3.0-4.0 mm from the midline.

Due to the area of bone that had to be left intact along the midline, it was impossible to insert the electrode vertically into much of the parahippocampal and hippocampal areas. Consequently, the electrode carrier was adjusted so that the electrode was at an angle of 30° from the vertical, in the coronal plane. Bilateral lesions were produced by passing RF current through the insulated insect pin electrode which, under visual guidance for the lateral and vertical co-ordinates, was inserted through the dura and into the hippocampal and parahippocampal areas at four positions in each hemisphere. The anterior-posterior co-ordinates, obtained from the atlas (Karten and Hodos, 1967), were A7.00, A6.00, A4.50, and A3.50, and at each placement the electrode was inserted approximately 1.5-2.5mm below the

surface of the brain.

On the basis of the calibration tests using egg albumen, and a pilot study using several pigeons, the following procedure, which was found to produce reliable lesions, was used throughout. The voltage output of the Grass Lesion Maker was initially set at minimum and the timer set for 60 seconds. At each electrode site a lesion was produced by switching on the RF current output and gradually increasing the voltage until a sudden drop in current occurred, as indicated on the milliammeter together with a concurrent rise in the voltage. This normally took 30 - 40 seconds and the average voltage and current values recorded immediately before the lesion was produced were approximately 30 volts and 30 mA. This procedure was carried out for each of the four electrode placements in the left hemisphere, and was then repeated in the right hemisphere. Any bleeding was controlled with sterile absorbable gelatine foam (Sterespon, May and Baker, Ltd., London) or with topical application of thrombin.

On completion of the lesions, small strips of Sterespon were packed into the trephine holes and the skull was dusted with antibiotic penicillin and sulphathiazole powder before carefully drawing together the skin flaps and closing them with five sutures. The wound was gently swabbed with a gauze moistened with alcohol and then the animal was removed from the stereotaxic instrument and placed in the recovery box, which was warmed by means of an Anglepoise lamp.

Three control groups of pigeons were used: a sham operated group underwent exactly the same procedure as described for the operated experimental group except that the lesioning electrode was not inserted into the brain and consequently no lesion was produced; a second control group was anaesthetised with Equi-Thesin and then allowed to recover as before; and the third group of animals were unanaesthetised and unoperated.

Histology

On the completion of the experiments the experimental animals were killed. They were given an overdose (8 mg/100 gms bodyweight, intrapectorally) of Nembutal (Abbott Laboratories) and perfused through the carotid artery with isotonic saline solution to clear the blood from the brain tissues, followed by 10% normal formol saline. The head was removed from the body, the calvarium removed, and the brain was allowed to fix in situ in 10% formol saline for 2-3 weeks. Before the brain was removed from the skull the head was mounted in the stereotaxic instrument, again using the Revzin adaptor and, with the aid of a No. 10 Swann-Morton scalpel blade inserted in the electrode carrier, the brain was blocked in the vertical plane of the instrument to ensure that, as far as possible, the histological sections would be in the same plane as those presented in the stereotaxic atlas of Karten and Hodos (1967).

The brain was then removed and placed in formalin for a further 3-4 weeks before being embedded in paraffin wax. Serial sections 10μ thick were cut on a base-sledge microtome and were then stained using mainly cresylecht violet and luxol blue, although occasionally haematoxylin and eosin were used instead. Every tenth section was saved and was mounted on a standard microscope slide under a cover slip using Canada balsam.

Reconstructions of the lesions were made using a series of drawings of coronal sections of the pigeon brain, extending from A8.50 to A3.25, and which were adapted from the stereotaxic atlas of Karten and Hodos (1967). Although drawings of the reconstructions for the individual pigeons are presented in the relevant chapters, a series of photographs the same size as the drawings is presented here in Figure 12 to show the placement and extent of a typical bilateral hippocampal lesion in the pigeon. A series of labelled drawings corresponding to the sections shown in the photographs in Figure 12 are presented in Figure 13.

ABBREVIATIONS

A	Archistriatum
Ad	Dorsal archistriatum
APH	Parahippocampal area
Av	Ventral archistriatum
Cb	Cerebellum
CDL	Dorsolateral corticoid area
CPi	Pyriform cortex
DMA	Dorsomedial nucleus of the anterior thalamus
E	Ectostriatum
Ep	Periectostriatal belt
GLv	Ventral portion of the lateral geniculate nucleus
HA	Accessory hyperstriatum
HD	Dorsal hyperstriatum
HISm	Hyperstriatum intercalatus suprema
Hp	Hippocampus
HV	Ventral hyperstriatum
IHA	Intercalated nucleus of the accessory hyperstriatum
LFB	Lateral forebrain bundle
MFB	Medial forebrain bundle
N	Neostriatum
NC	Caudal neostriatum
NF	Frontal neostriatum
NI	Intermediate neostriatum
OC	Optic chiasma
OPT	Principal optic nucleus of the dorsal thalamus
PA	Paleostriatum augmentatum

PP	Paleostriatum primitivum
Rt	Nucleus rotundus of the thalamus
S	Septal region
SGC	Stratum griseum centrale
SGF	Stratum griseum et fibrosum superficiale
TeO	Optic tectum
TSM	Corticoseptomesencephalic tract
V	Ventricle

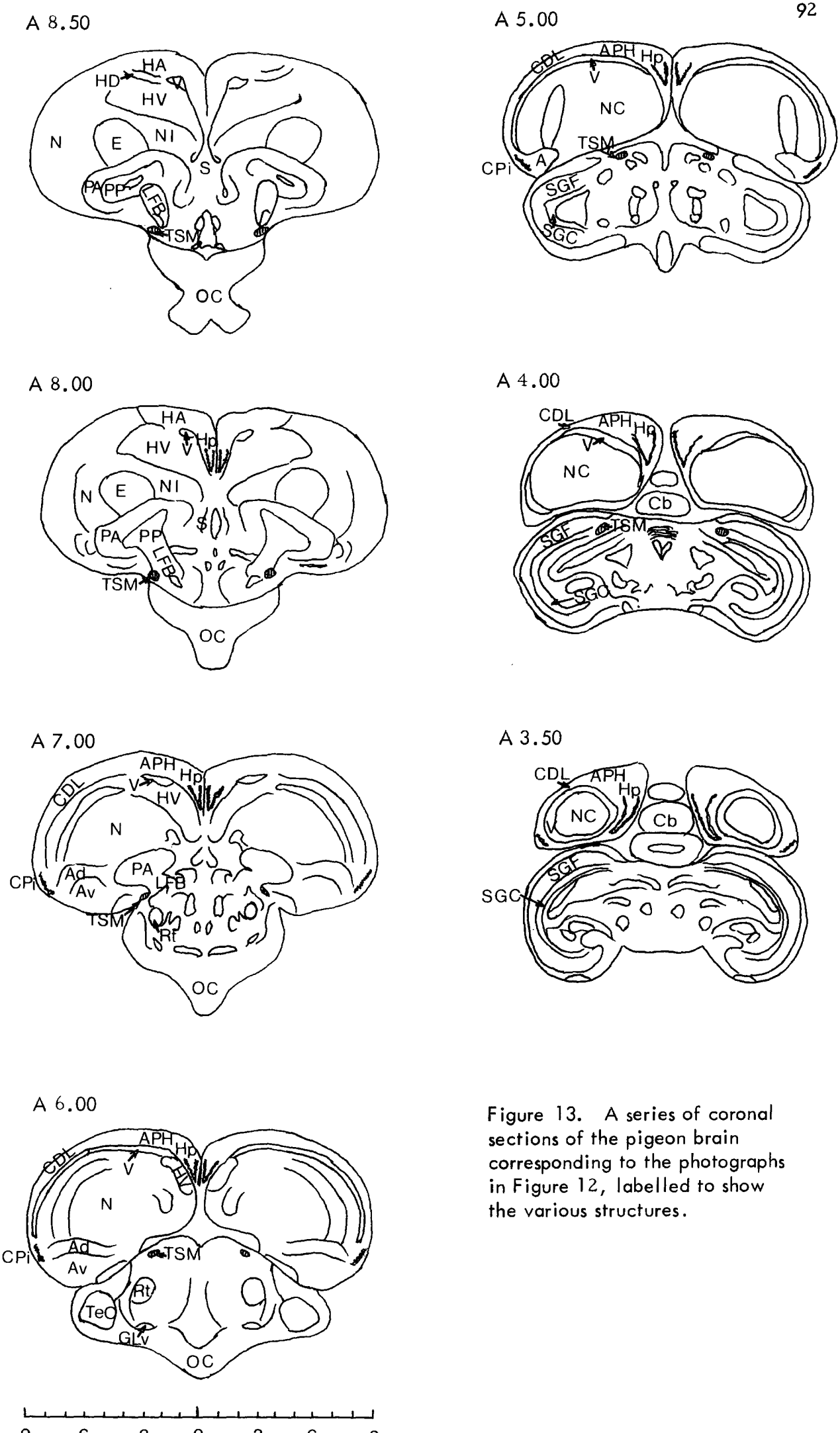


Figure 13. A series of coronal sections of the pigeon brain corresponding to the photographs in Figure 12, labelled to show the various structures.

Statistical analysis

In most of the experiments the data were analysed using a two-factor analysis of variance with repeated measures on one factor, the factors being lesion treatment x days (or reversals) (Keppel, 1973, p. 423). However, in the experiments reported in Chapters 6, 7, and 8 preliminary analysis of the data in each case incorporated a third factor, order of testing, since the three experiments were run as a balanced design (see Chapter 6). Trend analyses that were carried out used the coefficients for the linear components of the orthogonal polynomials (Keppel, p. 448), and a two-factor analysis of variance in which both variables were independent was also carried out on occasion (Keppel, p. 167). Other statistical analyses were carried out using either t tests or the Mann-Whitney U test (Siegel, 1956). The analyses of variance were carried by the computer at the University of Newcastle upon Tyne (NUMAC) using the Analysis of Variance/Covariance Processor (version of December 1975) available on the MTS Public Files.

From time to time, because of variability amongst animals across sessions, the problem of heterogeneity of variance was encountered. Although the differences in variance were never large, $\sqrt{x + 0.5}$ or log $(x + 1)$ transforms of the data were always considered, but because they did not substantially change the outcome of the analysis in each case, it was decided to use the untransformed data. This decision, it was felt, was confirmed by Keppel (1973) who states that "one of the most typical applications of the repeated-measures design is the learning experiment in which 'trials' is the independent variable. Under these circumstances it is quite likely to be the case that the assumptions of homogeneity of variance and of covariance are not met." and that "a great many experiments with repeated measures will lead to a questioning of the assumptions of homogeneous variances and covariances." (p. 464.). However, he also presents a cogent argument, after considering the various corrections that may be applied to data,

for the "practical, but less precise approach" of making no formal correction, since "statistical procedures are guides to aid us in guessing at which facts are "real" and which are not ... if a finding is interesting theoretically, we should not ignore its presence merely because it fails to exceed the critical F value or its corrected value." (pp. 466-467).

In most cases the normal, or control, groups were composed of pigeons that had undergone no surgical treatment whatsoever, those that had been anaesthetised, and those that had been anaesthetised and sham-operated. However, since preliminary analyses revealed no significant differences between these various subjects, they were treated as a single group of normal animals in each case.

General behaviour

No general behavioural effects of the bilateral hippocampal lesions were observed in any of the pigeons once they had recovered postoperatively. All pigeons were handled daily shortly after their arrival in the animal house, and no postoperative changes in activity or emotional response were detected when they were subsequently handled or fed, and no changes in food or water consumption were found to occur.

CHAPTER 3 The Acquisition and Reversal of a Visual Probability Discrimination

Introduction

The behavioural effects of hippocampal lesions in mammals are many and various, and although a short review of some of these effects was presented in chapter 1, more comprehensive reviews have been provided by a number of authors (e.g., Douglas, 1967; Kimble, 1968; Isaacson, 1974; and in particular, O'Keefe and Nadel, 1978). Amongst the effects are found impairments in the reversal of a simultaneous discrimination (Douglas and Pribram, 1966; Niki, 1966; Silveira and Kimble, 1968, Hirsh and Segal, 1972), but not the acquisition, unless it is a spatial task in which the animal is trained to the nonpreferred side (Samuels, 1972); greater resistance to extinction (Jarrard, Isaacson, and Wickelgren, 1964; Peretz, 1965; Douglas and Pribram, 1966; Henke and Bunnell, 1971); and poorer performance in a number of maze tasks, especially where place learning is involved (Kimble, 1963; Niki, 1966; Olton and Isaacson, 1968; Olton, 1972b; Plunkett, Faulds, and Albino, 1973; Sinnamon, Freniere, and Kootz, 1978).

Since the deficit appeared to be characterised by continued inappropriate responding in the presence of stimulus change and changes in reinforcement conditions the behaviour of hippocampal lesioned animals has most frequently been described as perseverative, and a number of hypotheses have been proposed to account for the deficits that occur. Thus, several investigators have suggested that hippocampal animals suffer from impaired response inhibition (Kimble and Kimble, 1965; McCleary, 1966; Altman, Brunner, and Bayer, 1973). However, a number of reports have been published in which it has been shown that hippocampal rats are capable of normal levels of response inhibition (Winocur and Salzen, 1968; Gaffan, 1972; Olton, 1972a; Winocur and Black, 1978; Plunkett and Faulds, 1979). An alternative hypothesis is that the hippocampus is involved in the inhibition of attention. Such a view was first proposed

by Douglas and Pribram (1966) in their model of limbic function, in which the hippocampus and the amygdala are each involved in an attention-directing system, the hippocampus being concerned with eliminating responses that lead to errors, and the amygdala acting to register responses that produce reward.

On the basis of this model Douglas and Pribram predicted that, without an error-evaluating mechanism, hippocampal lesioned animals would be impaired in a task involving inconsistent reinforcement. Such a task is a discrete-trials probability discrimination, in which responses to one stimulus are rewarded on a randomly distributed proportion of trials, while responses to the other stimulus are rewarded on the remaining trials. When trained on such a task without guidance, i.e. correction trials, normal animals invariably maximise their responses to the majority rewarded stimulus. Thus, Douglas and Pribram trained hippocampal lesioned monkeys on a visual probability discrimination using pattern stimuli, in which one cue was rewarded on 70% of the trials and the other was rewarded on the remaining 30% of trials (commonly referred to as a 70 : 30 probability task), and found that, compared with normal monkeys, the lesioned animals took significantly longer to maximise, i.e., to choose the more rewarded stimulus on 100% of trials, on both the acquisition and the reversal of the task. Similar deficits were reported by Stevens and Cowey (1973) and Nonneman, Voigt, and Kolb (1974) for hippocampal lesioned rats trained on a 70 : 30 spatial probability task. However, as O'Keefe and Nadel (1978) point out, in both of these experiments the animals had previously been trained on spatial discrimination or alternation tasks in the same maze, and the majority reward in the probability task occurred on the previously nonrewarded side, thereby making the probability discrimination effectively a complex form of spatial reversal task, on which hippocampal lesioned animals are generally impaired (e.g., Thompson and Langer, 1963; Niki, 1966; Hirsh and Segal, 1972). Evidence that supports this explanation comes from

another experiment by Stevens (1973a) in which he found that hippocampal rats, without extensive prior training, were actually superior to normal rats on an identical spatial probability task, although it should be pointed out that the rats were each given five pretraining trials to determine their preferred side and were then trained with their nonpreferred side rewarded on 70% of the trials. However, since this pretraining was minimal, it could be assumed to have had little effect on subsequent choice behaviour. Also, it is questionable whether five trials is sufficient to reliably determine a rat's position preference in a maze.

When given correction trials in a probability learning task, to ensure adequate exposure to the minority rewarded stimulus, rats no longer maximise, but neither do they match: their proportion of responses to the majority stimulus is less than 100% but is greater than the percentage of trials on which that stimulus is rewarded. Furthermore, birds respond less efficiently than rats, i.e., they choose the majority stimulus on a lower proportion of trials compared with rats. By analysing the pattern of errors made by rats trained with correction trials on probability learning experiments, Mackintosh (1970) has shown that the errors they made were not due to a temporary preference for the minority stimulus, but to a temporary failure to attend to the relevant cue. For example, when rats were trained on a 75:25 brightness probability discrimination, it was found that the proportion of errors that occurred on those trials on which the minority stimulus occupied the last rewarded position was significantly greater than chance, i.e., the rats reward-followed not on brightness (the relevant cue), but on position (the irrelevant cue). Thus, rats "do not select the minority stimulus because of a (momentary) preference for it, but because they select a particular position and the minority stimulus happens to be in that position" (Mackintosh, 1970, pp. 180-181). In addition, Mackintosh and Holgate (1968) found that rats learned the reversal of a 75:25 brightness probability task more slowly than they learned the reversal of a 100:0 brightness discrimination,

because the inconsistently reinforced animals were more likely to develop position habits during reversal, and they concluded that this was due to their having failed to learn to attend adequately to the relevant cue. Mackintosh (1969) has further shown that, like rats, chicks trained on a 75:25 brightness probability task also do not reward-follow on brightness, but on position. Hence, Sutherland and Mackintosh (1971) have argued that the reduction in efficiency on probability tasks when correction trials are given is due to lapses of attention to the relevant cue, and that birds are more prone to such lapses of attention and hence are more likely to respond to the minority stimulus.

If hippocampal lesions affect attentional processes, as Douglas and Pribram have suggested, then hippocampal lesioned birds ought to perform less efficiently than normal birds on a probability task with guidance, since their impaired attention should make them more likely to respond to the irrelevant cue, and therefore to the minority rewarded stimulus. Similarly, they ought to be impaired on the reversal as well, so that at asymptote they would be responding to the majority stimulus on a lower proportion of trials than normal birds.

However, Samuels (1972) has pointed out that, although there is evidence to support the notion that hippocampal animals are impaired in their ability to inhibit attention, there appears to be a greater emphasis in the literature on spatial, as opposed to visual, deficits, particularly on reversal tasks, and she has therefore suggested the possibility of a specifically spatial impairment rather than a general inhibitory deficit. And indeed, from the results of her experiments on transfer effects in the acquisition and reversal of simultaneous spatial and brightness discriminations, she concluded that the hippocampal deficit could not be explained either in terms of impaired response suppression or of impaired attentional processes. Instead, she claimed her results suggested the importance of a spatial factor, since the hippocampal

rats were impaired on the acquisition of, or the transfer from a visual task to a spatial task if the positive stimulus was the non-preferred side, but corresponding deficits did not occur on either the acquisition of a visual task, or on the transfer to it from the spatial task, when the nonpreferred brightness was the correct stimulus.

Other evidence that supports the proposal that the hippocampus plays a particular role in normal spatial function has been provided by the experiments of Olton and Isaacson (1968), Mahut (1971, 1972), Mahut and Zola (1973), Plunkett, Faulds, and Albino (1973), and Sinnaman, Freniere, and Kootz (1978) (see also O'Keefe and Nadel, 1978). As Samuels (1972) had found in hippocampal rats, Mahut, and Mahut and Zola found that monkeys with hippocampal lesions or with transection of the fornix were impaired on spatial reversals but not on visual or object reversals. Furthermore, it has sometimes been shown that hippocampal rats are also impaired on the acquisition of a simultaneous spatial discrimination, but it appears that a deficit only occurs when the animals are trained on their nonpreferred side, because they seemingly have great difficulty in changing preferred responses, especially spatial ones, and therefore are likely to make long sequences of inappropriate responses (Means, Woodruff, and Isaacson, 1972; Samuels, 1972).

If the avian hippocampus is similarly involved in spatial function, hippocampal lesioned birds ought to be similarly impaired on spatial reversals, but not necessarily on visual reversals. Also, they should show a learning deficit on the acquisition of a spatial discrimination if trained against their preferred side. But in particular, because of impaired spatial ability, and difficulty in changing preferred responses, when trained on either the acquisition or the reversal of a visual probability discrimination with guidance, hippocampal lesioned birds ought not to reward-follow on position to the same extent as normal birds. Consequently, they should either make fewer responses to the minority stimulus and correspondingly more to the majority stimulus; or they should adopt a position habit, and thereby respond less to the majority

stimulus than normal birds, possibly responding to it only at chance level for a long time. However, since birds are noticeably more visual animals than rats are (for example, see Sutherland and Mackintosh, 1971, pp. 281 and 438), it is possible that they would be more likely to respond consistently to the visual stimulus than to adopt a position habit. Indeed, Sutherland and Mackintosh (1971) have suggested that "... just as rats do not, for example, have to learn to attend to spatial cues, so birds may not have to learn to attend to simple visual cues" (p. 438). Evidence that supports this proposal comes from the failures to obtain an overlearning reversal effect (ORE) in rats trained on a spatial discrimination or in birds trained on a simple visual problem.

It is therefore predicted that, if hippocampal lesions impair spatial functions in birds as in rats, then hippocampal lesioned birds trained on a simple visual probability discrimination with correction trials ought to reward-follow on position less than normals, on both acquisition and reversal; and because of the particular salience of the relevant (visual) cue for birds, the lesioned animals should respond more consistently to the majority rewarded stimulus than normal birds.

A further effect that has often been found to occur in mammals with hippocampal lesions is an increased resistance to extinction (see Chapter 1, p. 52). Thus, extinction deficits have been reported in rats in a runway (Jarrard, Isaacson, and Wickelgren, 1964; Winocur and Mills, 1969), in a Y-maze (Kimble and Kimble, 1970), and in an operant chamber (Rabe and Haddad, 1968; Henke and Bunnell, 1971), in cats trained in a WGTA (Peretz, 1965; Brown et al, 1969), and in monkeys trained in an operant chamber (Douglas and Pribram, 1966). Various explanations have been proposed to account for these effects, and they include a loss of response inhibition (Brown et al, 1969), increased response perseveration (Rabe and Haddad, 1968), and impaired attention-shift behaviour (Douglas and Pribram, 1966; Kimble and Kimble, 1970).

However, there have also been some reports in which rats with hippocampal lesions were not impaired in extinction (Schmaltz and Isaacson, 1967, and Nonneman et al, 1974: operant task; Nadel, 1968: conditioned suppression task; Ackil, Mellgren, Halgren, and Frommer, 1969: two-way active avoidance task), and recently O'Keefe and Nadel (1978) have suggested that extinction deficits occur mainly in runway and maze situations, and rarely in avoidance tasks, classical conditioning situations, or operant tasks. They have argued that, in the normal, intact animal, place hypotheses play an important part in the learning and extinction of responses in a maze or a runway, and that, because of their flexibility, and because exploratory behaviour is readily elicited by a change in conditions, place hypotheses are easily extinguished. On the other hand, animals with hippocampal lesions have to rely on the use of the non-hippocampal guidance and orientation hypotheses, which are not flexible and show persistence. However, because all animals have very little opportunity to use place hypotheses in an operant chamber, both normal and hippocampal animals should have to rely on guidance and orientation hypotheses to a similar extent, and therefore should perform similarly.

Thus, a group of hippocampal lesioned pigeons and a group of normal pigeons were trained, with correction trials, on the acquisition and reversal of a 70 : 30 colour probability discrimination. The birds were each given a total of 2000 trials which, it was expected, was more than adequate to allow them to reach asymptotic levels of performance, and then received a further 2000 trials on the reversal of the probability task. In order to compare the extinction performance of hippocampal and normal pigeons in an operant chamber, they were given three extinction sessions following the completion of each stage of the experiment.

Method

Subjects

Ten experimentally naive pigeons, maintained at 80% of their ad lib bodyweights and given free access to water in their home cages, were used in this experiment. Five of the pigeons were either sham-operated or unoperated control subjects and five were given bilateral hippocampal lesions.

Apparatus

A standard two-key operant chamber was used which was lit by a white houselight and the keylights could be either red, green, or white.

Procedure

Pretraining

Preoperatively, all ten pigeons were habituated to the apparatus, food-magazine trained, and hand-shaped to peck at either key regardless of the colour of the keylight. On the following day, in order to give them equivalent training on both keys, they were each given a single pretraining session of 60 trials, 30 on each key. All three keylight colours were used so that, on each key each of the three colours was presented for a total of ten trials, and the order in which they were presented was determined by Gellerman sequences. On each trial only one key was lit, and the order in which the left and right keys were presented was also determined by Gellerman sequences. For the first twenty pretraining trials a single response (i.e., a CRF schedule) on the lighted key resulted in 3 secs access to food, during which period the keylight was turned off. At the end of reinforcement the appropriate keylight came on to signal the start of the next trial. The response requirement was then changed to two responses (FR2) on the twenty-first trial, and finally to three responses (FR3) on the forty-first trial.

Three days after the completion of pretraining a random half of the animals

underwent surgery, and the remaining animals were operated upon the following day. All pigeons were given approximately 14 days for postoperative recovery, and they were then given a further pretraining session of 60 trials, which were identical to the first pretraining session except that the response requirement was FR 3 throughout. This postoperative session was to ensure that the pigeons responded readily to either key whenever it was presented, irrespective of its colour, and to the food hopper when it was operated. It was also intended as a measure of postoperative change on a simple operant task.

Training

Training in the probability discrimination began on the day following the post-operative pretraining session. Both keys, one of which was red and the other green, were presented simultaneously on each trial. A 70:30 reinforcement schedule was used, and during the acquisition stage responses to the green key were reinforced on 70% of the trials, responses to the red key being reinforced on the remaining 30% of trials. The spatial presentation of the two colours, and the order in which they were to be reinforced, were determined by specially modified Gellerman sequences which included the following restrictions: in each block of 10 trials responses to the green key would be reinforced on 7 occasions, and to the red key on the remaining 3 occasions; in each daily session no more than four consecutive reinforcements would be available following responses to the green key, and no more than two consecutive reinforcements following red key responses; and finally, responses to a particular key would not be reinforced on more than three consecutive trials.

At the start of each training session the houselight and both keys were lit. FR 3 on the correct key (the key presenting the colour which was scheduled to be reinforced) switched off both keylights and was reinforced by 3 secs access to food. FR 3 on the incorrect key also switched off both keylights, but in place of reinforcement

the houselight was switched off for 3 secs timeout (TO). Following either reinforcement or TO there was a 5 secs intertrial interval (ITI), during which the keys remained off and inoperable, and then the keylights came on again for the start of the next trial. Responses on the two keys were counted separately so that, whether or not the trial was reinforced depended on which key first accumulated three responses, and during the ITI the two predetermined counters used to control the FR 3 schedule on the two keys were automatically reset.

A correction trial procedure was used throughout in which, following an incorrect response the trial was repeated, but only the correct key was presented, the other remaining unlit and inoperable. Thus, a correction trial was always reinforced. Each animal was run daily until it had obtained 100 reinforcements, and acquisition training was continued for 20 days. On each of the three following days a $\frac{1}{2}$ hour extinction session was given, in which the procedure was basically the same as that used in acquisition, except that neither reinforcements nor correction trials were given.

On the day after the third extinction session reversal training began in which the procedure was identical with that used in acquisition, except that the red key was now reinforced on 70% of the trials. All pigeons were given 20 days of reversal training, and each daily session again continued until 100 reinforcements had been gained. On each of the three days following the completion of the reversal stage of the experiment a $\frac{1}{2}$ hour extinction session was given, the procedure being the same as that used before. On the completion of each daily acquisition, reversal, or extinction session both keylights were automatically switched off and the keys became inoperable, although the houselight remained on.

Four stimulus presentation sequences were prepared, each of which contained ten blocks of ten trials. These are presented in the Appendix. They were punched on to paper tapes which, when fed into a small paper tape reader unit (manufactured by

Tally, Ltd., London) which was built into an electromechanical programming module, were used to control the sequence of events presented to the pigeons. Electromechanical counters were used to record the total trials in each session on which FR 3 was completed on left, right, green, and red keys.

Surgery

Full details of the surgical procedures involved are presented in Chapter 2.

Results

Histology

Reconstructions of the lesions of the five hippocampal pigeons in this experiment are shown in Figure 14. In four of the pigeons (Nos. 3, 6, 9, and 15) the lesions extended, variously, from A 8.00 to A 3.50, the lesion in any one animal being at least 3-4 mm in extent in the anterior-posterior plane. It can also be seen that the lesions in each pigeon were very approximately equivalent in the two hemispheres. Most damage occurred in the region which Huber and Crosby (1929) called the hippocampus pars dorsalis, and which is now generally regarded as the avian homologue of the mammalian cornu ammonis, or hippocampus proper (see Table 1 in Chapter 1, and pp. 60-63). Smaller amounts of damage occurred in the hippocampus, the presumed homologue of the mammalian fascia dentata, and also in the parahippocampal area, and in most of these pigeons very minor invasion of the ventral hyperstriatum (HV) also occurred. In the fifth pigeon (No. 4) it can be seen that damage occurred only unilaterally, and the lesion, besides being fairly small, involved only a minimal region of the parahippocampal area, and part of the dorsolateral corticoid area (CDL). Thus, in this animal no hippocampal damage occurred in either hemisphere, and it can therefore be regarded as equivalent to a sham-operated pigeon.

Pretraining

All pigeons readily adapted to the apparatus and to feeding from the food hopper

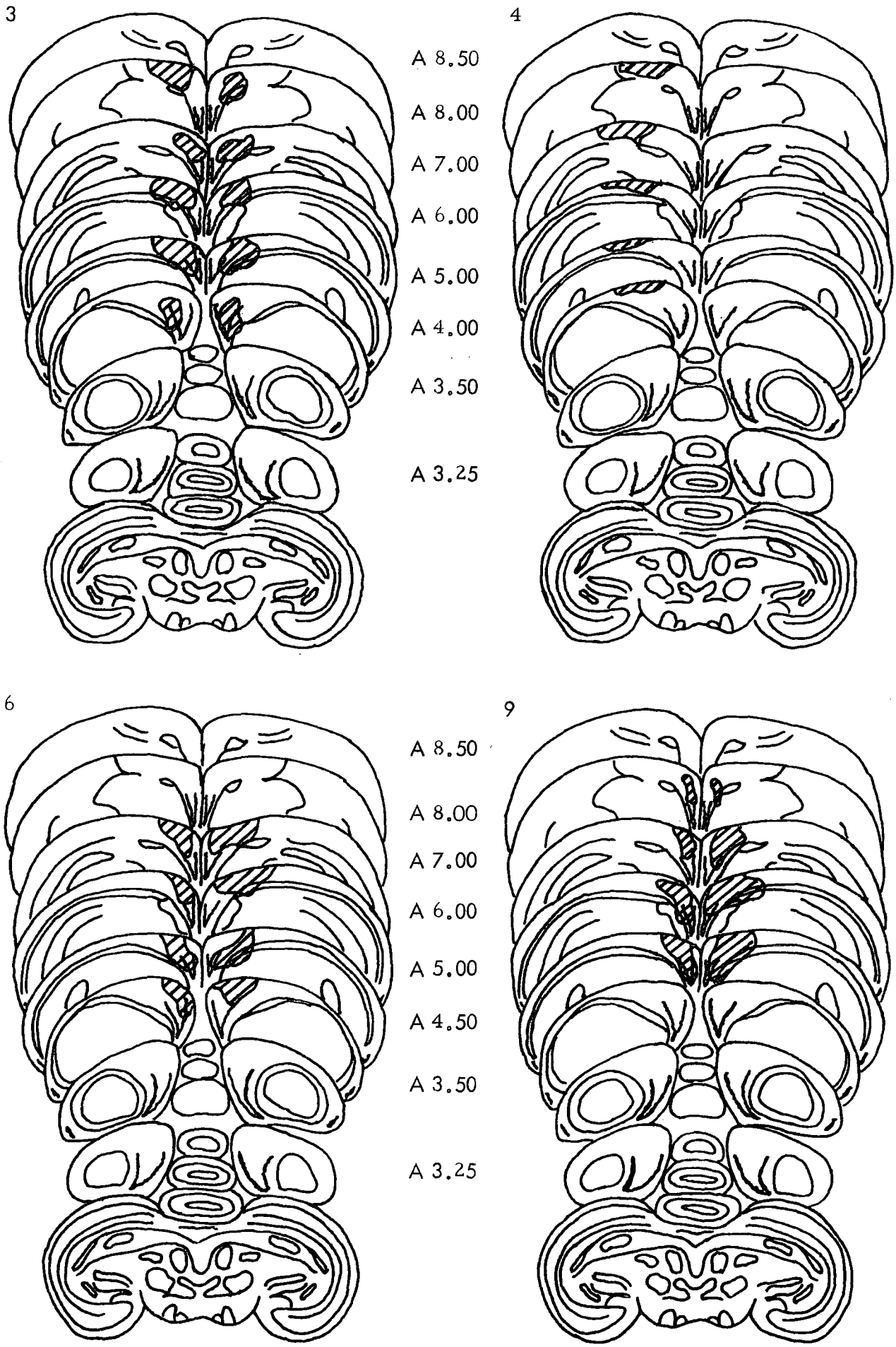


Figure 14. Reconstructions of the hippocampal lesions in each of the five experimental pigeons, based on the stereotaxic atlas of Karten and Hodos (1967).

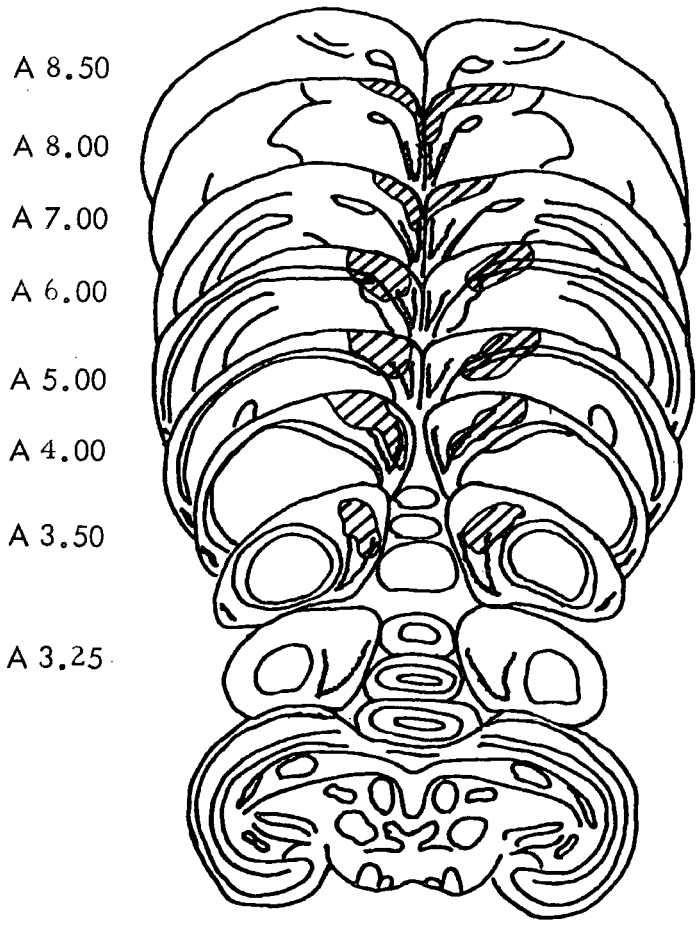


Figure 14 (contd.)

whenever it was presented. They all learned fairly quickly first to peck at a key, and then to peck at either key regardless of its colour. In the first, preoperative, pretraining session they all quickly transferred from CRF to FR 2 to FR 3. In the second session, which was carried out postoperatively, all pigeons responded very much as they had done preoperatively, and no postoperative changes were observed.

Training

Despite correction trials, two pigeons, one from each group, began to adopt a marked position preference during acquisition training, and towards the end of acquisition and throughout the whole of reversal training they were both responding to their preferred position on 90% - 100% of trials. The animal in the experimental group was pigeon No. 4, the bird which had not received any hippocampal damage and which was therefore equivalent to an operated control animal. Consequently, the data from these two pigeons were omitted from the analysis and the number of pigeons in each group was therefore reduced to four. In each of the measures presented here, in both acquisition and reversal, correction trials were not included in the data analysis.

Acquisition

The numbers of responses to the majority rewarded colour, i.e., green, were calculated as a mean percentage in each daily block of 100 trials for each group, and are presented in Figure 15. From this it can be seen that the percentage choice of the majority stimulus by both groups was a little above chance level on the first day. For the normal group this score increased fairly rapidly, initially to 81% on the third day, but was subsequently maintained at approximately 70% until the end of the acquisition stage (overall mean, 68.6%; mean over days 6-20, 68.0%. In contrast, the hippocampal pigeons showed a gradual increase in their choice of the majority stimulus, which they maintained throughout acquisition, achieving a mean score of 91% on the final day (overall mean, 71.2%). These data were subjected to an analysis of variance

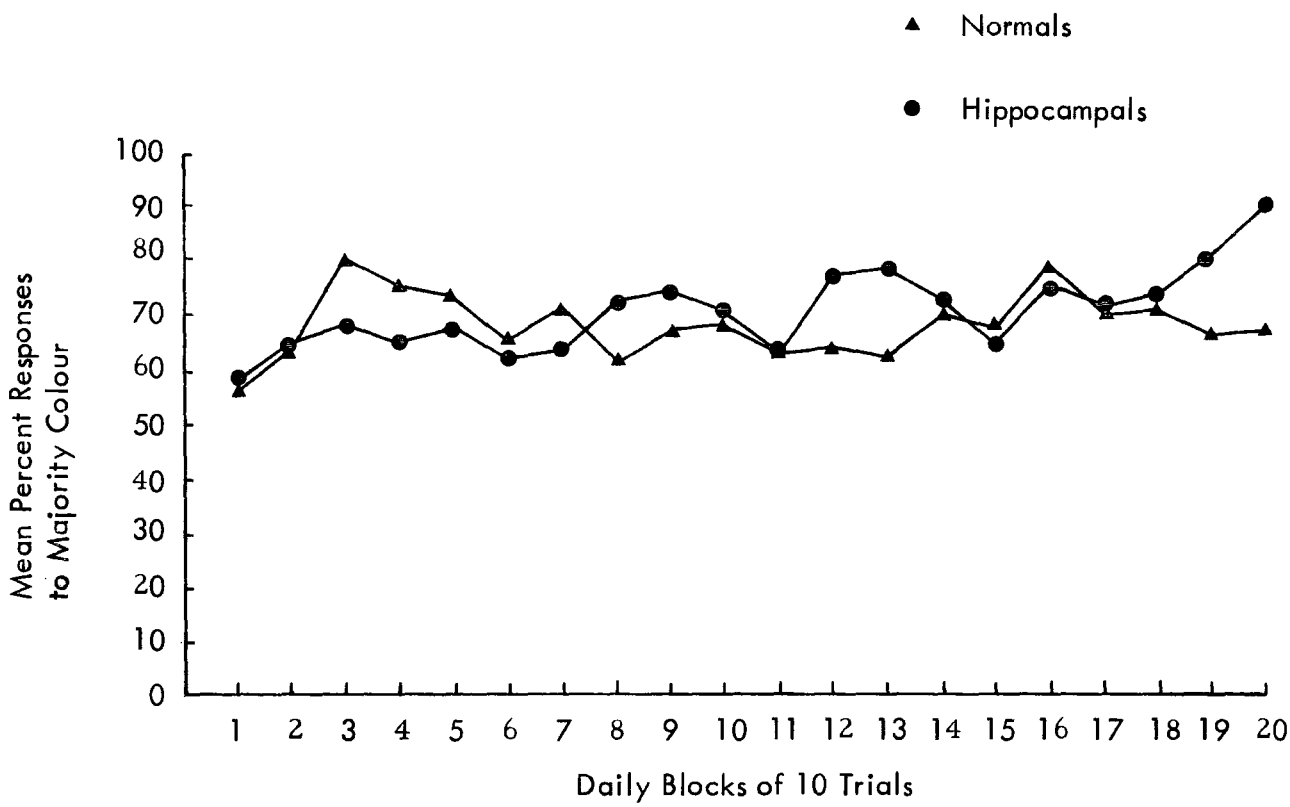


Figure 15. Responses to the Majority Colour in Acquisition.

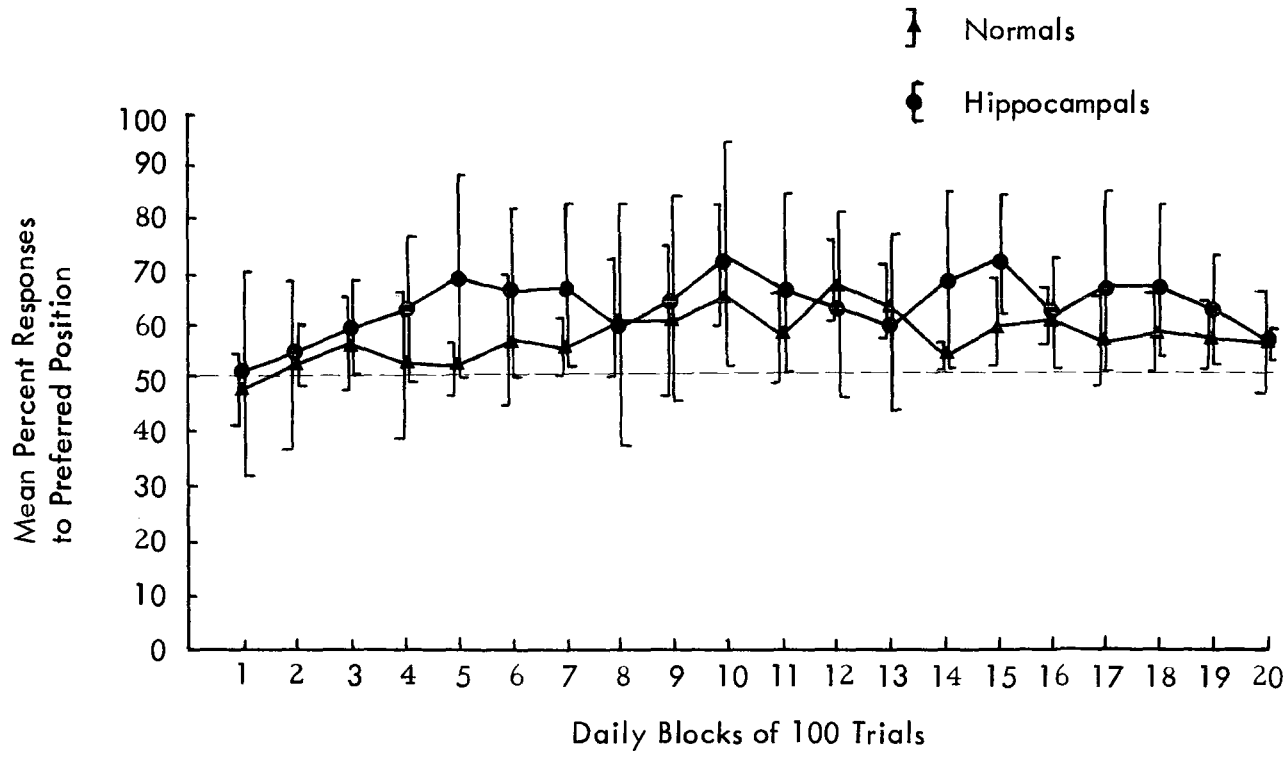


Figure 16. Responses to the Preferred Position in Acquisition

which showed that, overall, there was no significant difference between the two groups ($F(1,6)=0.22$, $p=0.66$), but that there was a significant increase in the choice of the majority colour over days ($F(19,114)=2.58$, $p=0.001$) and the interaction with the groups over days was significant ($F(19,114)=2.15$, $p=0.007$). A trend analysis using orthogonal polynomial coefficients (Keppel, 1973, pp. 448-454) was then carried out which showed that there was a significant linear trend over the 20 days ($F(1,6)=13.41$, $p<0.025$) and that the interaction of the linear trend components over days for the two groups was equally significant ($F(1,6)=9.45$, $p<0.025$). Finally, separate trend analyses were carried out for each group, and there was found to be a significant linear trend over days for the hippocampal group ($F(1,3)=25.39$, $p<0.025$), but not for the normal group ($F(1,3)=0.16$, $p>0.25$). Thus, the normal pigeons fairly quickly adopted a matching level of performance, which they maintained until the end of acquisition. On the other hand, while the hippocampal group showed a significant trend towards maximising, the differences between the two groups appear to be rather small, and since the largest difference between the groups occurs on the final two days, it would seem unwise to place too much emphasis upon these differences in acquisition.

Each pigeon's preferred position was determined simply by counting the total numbers of left and right key responses that they each made during acquisition, and the side to which the majority of responses were made was taken to be the preferred position. Then, for each group, the mean percentage response to the preferred position were calculated for each of the 20 days. These data are presented in Figure 16, and from this it can be seen that both groups began by responding at chance level to position. Subsequently, these responses increased so that, overall, both groups were responding to their preferred position on approximately 55% - 65% of trials, the hippocampal pigeons making slightly more position responses than the normal pigeons (overall means: hippocampal group, 64.2%; normal group, 58.2%). An analysis of variance revealed

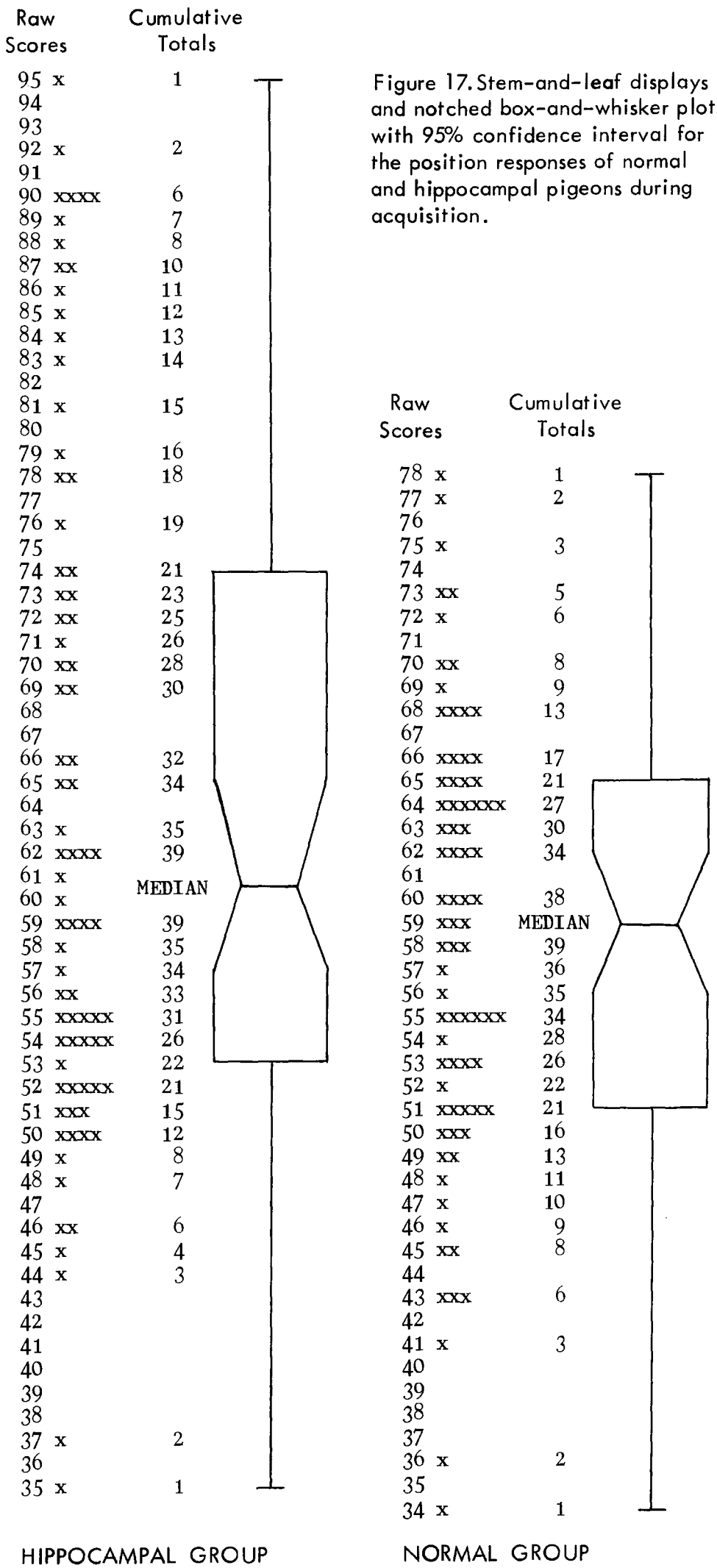


Figure 17. Stem-and-leaf displays and notched box-and-whisker plots with 95% confidence interval for the position responses of normal and hippocampal pigeons during acquisition.

that there were no significant differences between the two groups ($F(1,6)=1.75$, $p=0.19$), the effect over days was not significant ($F(19,114)=1.31$, $p=0.23$), and neither was the interaction between the groups over days ($F(19,114)=0.60$, $p>0.89$).

In order to determine whether or not the position response scores differed from chance, stem and leaf displays and box-and-whisker plots (Tukey, 1977; McGill, Tukey, and Larsen, 1978) were drawn for each of the two groups and are presented in Figure 17 (the data summaries and calculations are in the Appendix). These provide a particularly convenient means of displaying raw data together with measures of central tendency and of dispersion. Thus, it can be seen from these displays that the position responses of both groups were above chance level, the scores of the hippocampal animals showing a slightly greater deviation from chance than those of the normal animals. Standard deviations of the mean daily scores for each group are also plotted on Figure 16, and these data lend support to the view that the hippocampal pigeons were responding to their preferred position mostly at above chance level at least over days 5 - 20. In general, the normal pigeons also appear to have been responding to position at above chance level, although less reliably so.

Extinction 1

The total numbers of responses made by each pigeon during the three extinction sessions are shown in Figure 18A. Although it can be seen from this that the hippocampal pigeons made more responses than the normal pigeons on each of the three days, an analysis of variance showed that none of these differences was significant ($F(1,6)=5.41$, $p=0.06$) but there was a significant reduction in responding over the three days by both groups ($F(2,12)=16.28$, $p=0.0004$), although, as expected from the graph, there was not a significant interaction between the groups over days ($F(2,12)=0.29$, $p=0.76$).

Because of the differences in the total numbers of responses made by each pigeon, the majority colour responses were calculated as a percentage of the total daily responses

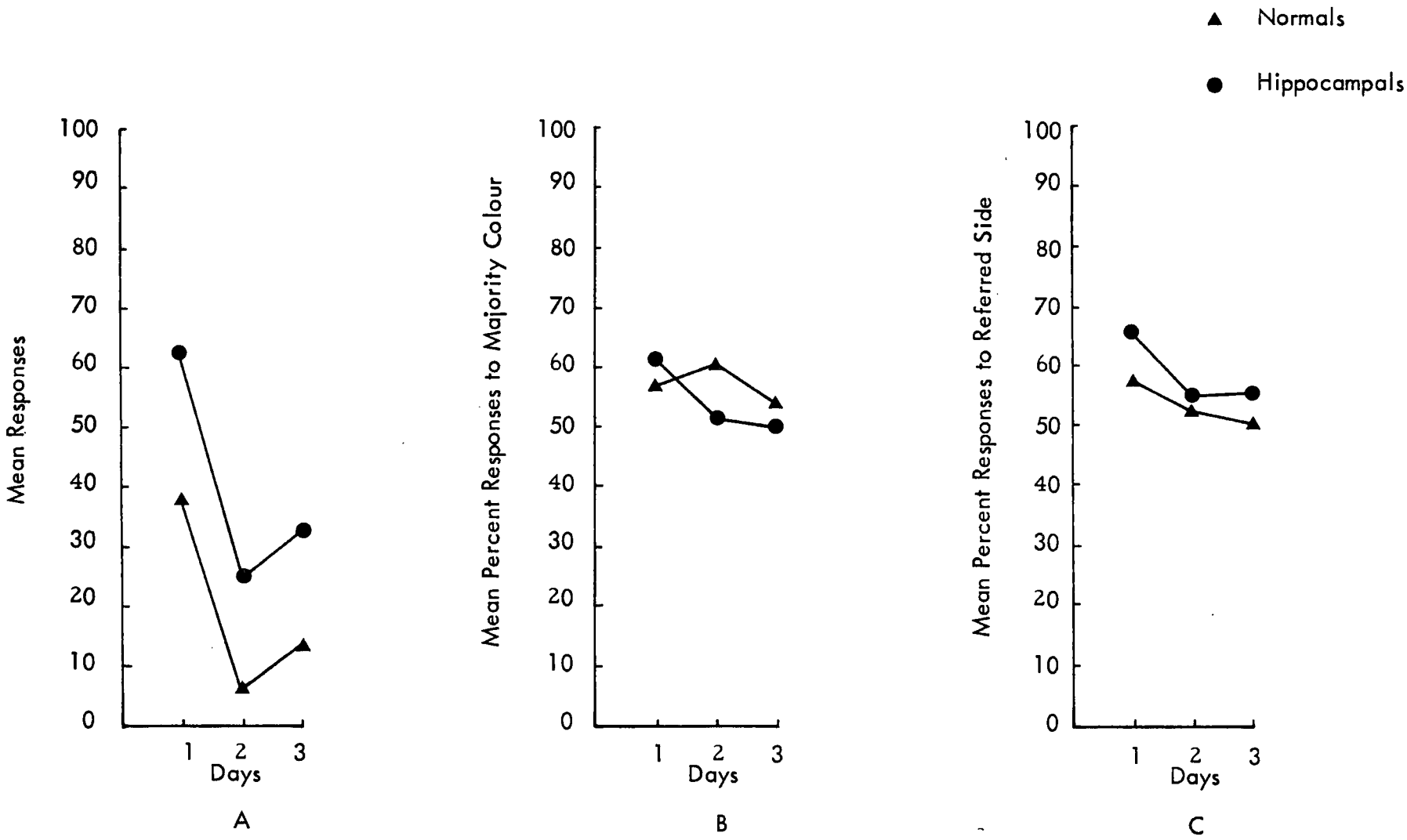


Figure 18. Total Responses and Mean Percent Responses to Majority Colour and to Preferred Position in Extinction following Acquisition of a 70:30 Colour Probability Discrimination.

for each animal, and the group data are presented in Figure 18B. From this it can be seen that the responses of both groups to the majority colour declined from approximately 60% on the first extinction session to chance level on the third day. An analysis of variance confirmed that there were no significant differences overall between the two groups ($F(1,6) = 0.08$, $p = 0.78$), the effect over days was not significant ($F(2,12) = 0.51$, $p = 0.62$), and the interaction between the two groups over days was not significant ($F(2,12) = 0.42$, $p = 0.67$).

Finally, the responses to the preferred position, also calculated as percentages, were analysed. These scores are presented in Figure 18C and this shows that both groups reduced their position responses to about chance level over the three extinction sessions. The analysis of variance again confirmed that, overall, there were no significant differences between the two groups ($F(1,6) = 0.42$, $p = 0.54$), that there was not a significant reduction in responses to the preferred position over days ($F(2,12) = 1.99$, $p = 0.18$), and that the groups x days interaction was not significant ($F(2,12) = 0.18$, $p = 0.84$).

Reversal

During reversal the majority rewarded colour was now red, and the mean percentages of responses to this colour are presented in Figure 19. This shows that both groups responded to the red key at about chance level on the first day. The normal group then gradually increased their choice of the majority stimulus to approximately 60%, a level of response which they maintained until the end of reversal. In contrast, the hippocampal pigeons progressively increased their choice of the majority colour over the 20 days so that, on each of the last six days in reversal they were responding to the red key on 90% or more of the trials. As before, these data were subjected to an analysis of variance, which showed that the hippocampal pigeons made significantly more responses to the majority rewarded colour than the normal pigeons ($F(1,6) = 16.82$, $p < 0.007$), and that the effect over days ($F(19,114) = 7.04$, $p < 0.00005$) and the interaction of the groups over days ($F(19,114) = 4.34$, $p < 0.00005$)

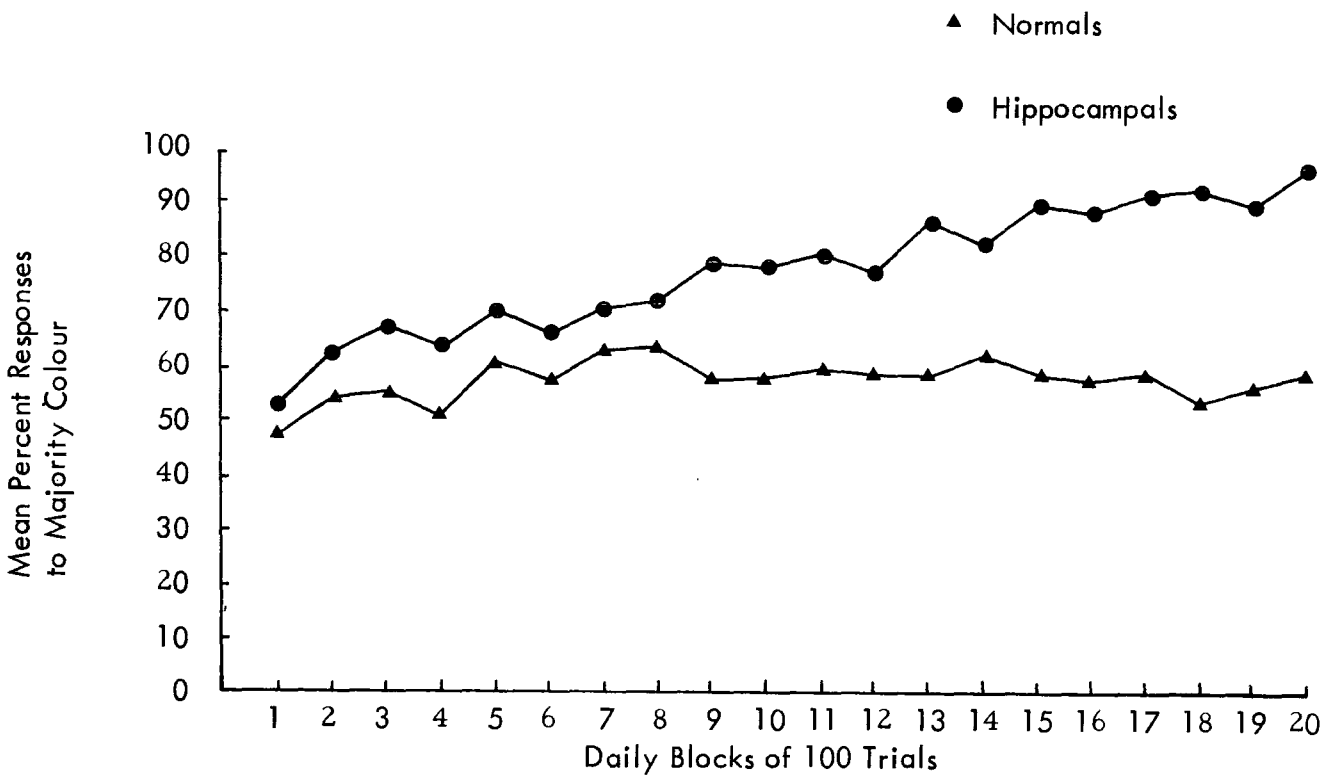


Figure 19. Responses to the Majority Colour in Reversal

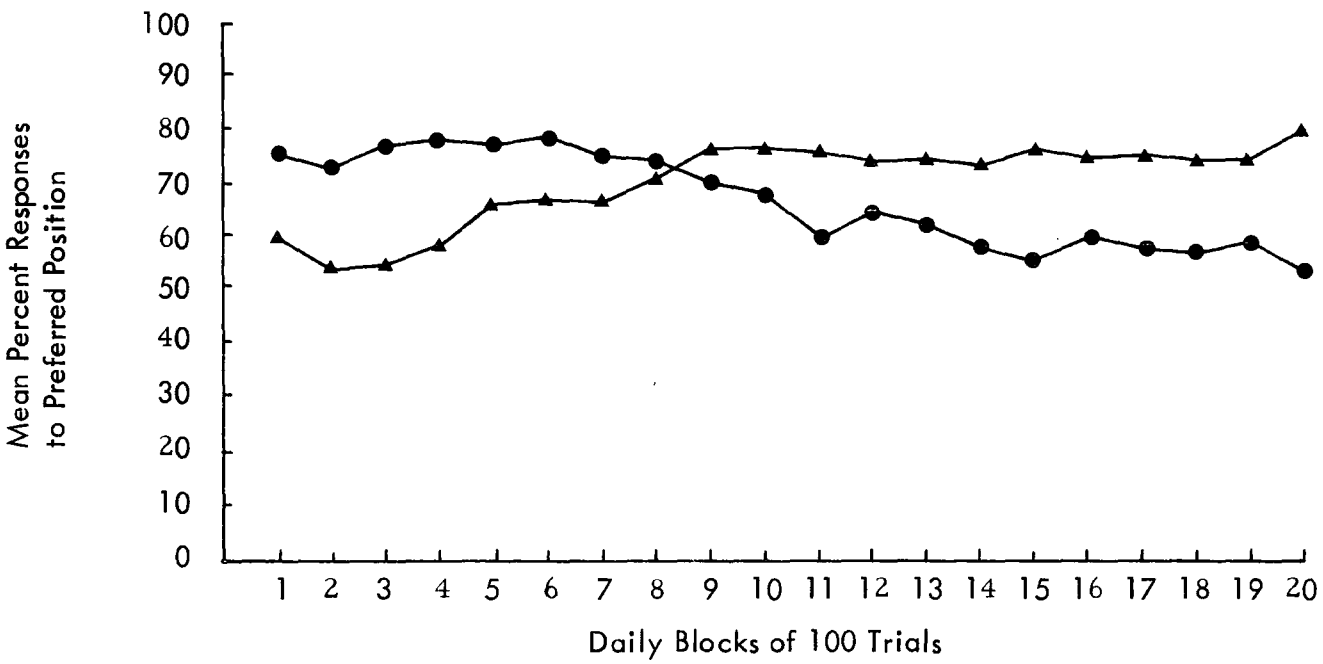


Figure 20. Responses to the Preferred Position in Reversal

were both highly significant. A trend analysis was also carried out, which revealed a significant linear component in the effect over days ($F(1,6) = 33.19$, $p < 0.01$), and a significant interaction between the linear trend components ($F(1,6) = 21.07$, $p < 0.01$). Finally, separate trend analyses for the two groups showed that there was a significant linear trend in the majority colour responses over days for the hippocampal pigeons ($F(1,3) = 31.16$, $p < 0.025$), but not for the normal pigeons ($F(1,3) = 2.44$, $p > 0.10$). It can be seen, therefore, that over the first five days of reversal training the normal pigeons increased their choice of the majority stimulus to approximately 60% and then maintained this level of performance for the next 15 days, whereas the hippocampal pigeons showed a progressive increase over days in their choice of the majority rewarded stimulus and a clear trend towards maximising.

The position preferences in reversal were determined for each pigeon as before, and the mean percent position responses for each group are shown in Figure 20. By comparing this with Figure 17 it can be seen that the normal pigeons made approximately the same proportion of position responses at the beginning of reversal as they did at the end of acquisition, i.e., about 60%. But, following a brief decline towards chance level over the next two days, they increased their position responding to approximately 75% by the ninth day of training, which they then proceeded to maintain until the end of reversal training. In contrast, the hippocampal pigeons, who also had made about 60% position responses at the end of acquisition, began reversal with a marked position preference, which they maintained over the first eight days and then progressively gave up, so that by day 20 they were responding to position at about chance level. An analysis of variance showed that there were no significant differences overall between the two groups ($F(1,6) = 0.22$, $p = 0.65$), and that the effect over days was not significant ($F(19, 114) = 0.80$, $p = 0.70$), but, as expected, the interaction between the two groups over days was highly significant ($F(19,114) = 6.23$, $p < 0.00005$). A trend analysis was carried

out, which confirmed that the linear component of the trend over days was not significant ($F(1,6)=0.14$, $p \gg 0.25$), but that there was a significant linear component in the groups x days interaction ($F(1,6)=21.25$, $p<0.01$). Separate trend analyses were then carried out for the two groups, and they showed that there was a significant linear trend over days for the hippocampal group ($F(1,3)=28.72$, $p<0.025$) but not for the normal group ($F(1,3)=5.74$, $0.10>p>0.05$).

Extinction 2

The mean responses made by the two groups on each of the three extinction sessions are shown in Figure 21A, and it is clear from this that the normal pigeons made more extinction responses than the hippocampal pigeons on the first day, although there appears to be little difference overall between the two groups. This was confirmed by an analysis of variance, which showed that the difference in scores between the two groups was not significant ($F(1,6)=0.09$, $p=0.76$), and that there was not a significant groups x days interaction ($F(2,12)=1.75$, $p=0.21$), but there was a significant reduction in responses over the three days ($F(2,12)=9.60$, $p=0.003$).

As before, the responses to the majority colour were calculated as a percentage of the total daily responses for each pigeon, and the mean values for the two groups are shown in Figure 21B. Compared with the last few days of reversal, the hippocampal pigeons have shown a reduction in their choice of the majority stimulus from a near maximising level to a matching level, although of course these latter responses were not rewarded. In contrast, the normal pigeons have, if anything, marginally increased their majority colour responses over their score during the last few days of reversal. Nevertheless, throughout the three extinction sessions the hippocampal pigeons maintained a higher level of response to the majority colour than the normal pigeons. An analysis of variance confirmed that the hippocampal group made significantly more red key responses than the normal group ($F(1,6)=9.21$, $p=0.023$), and that neither the

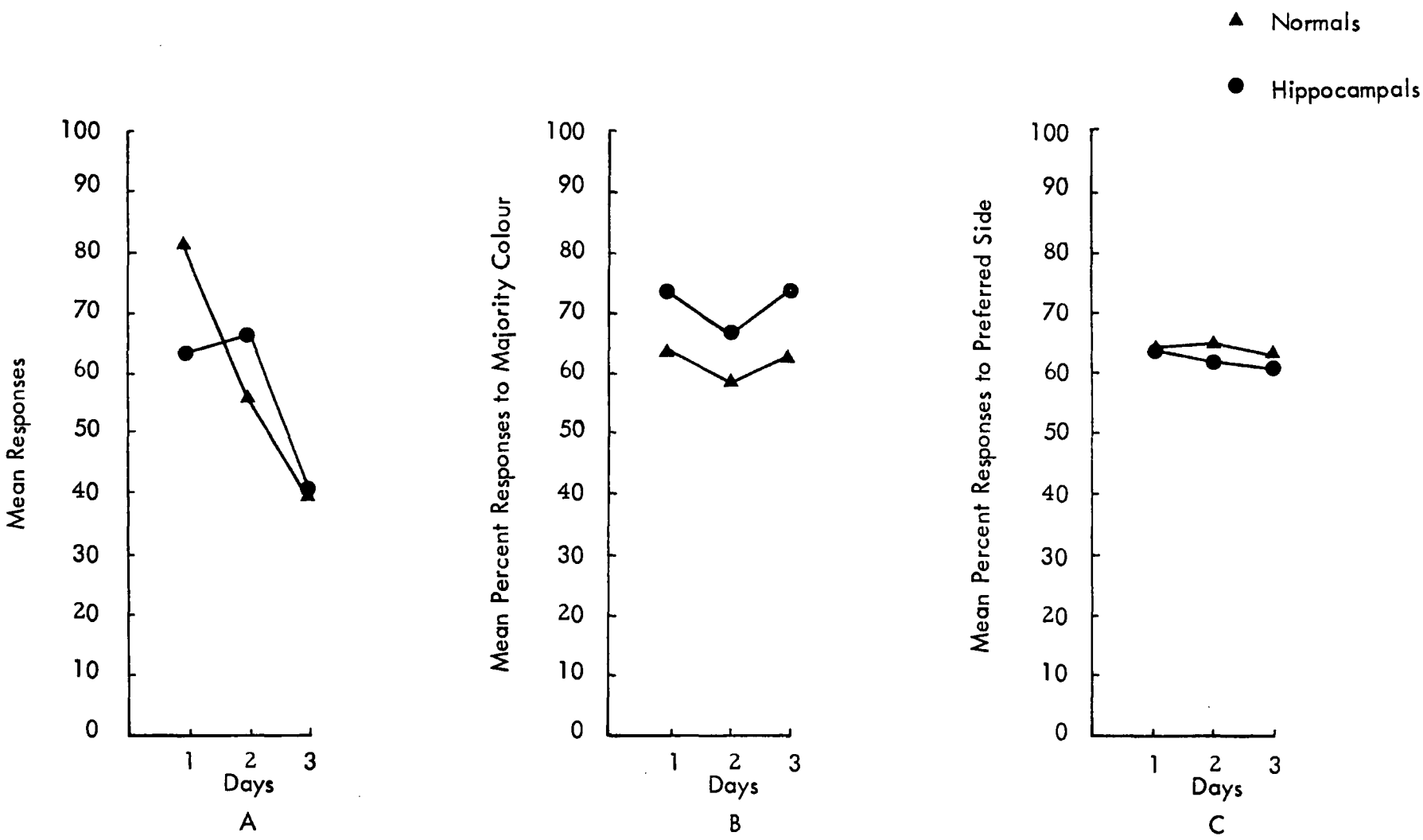


Figure 21. Total Responses and Mean Percent Responses to Majority Colour and to Preferred Position in Extinction following Reversal of a 70:30 Colour Probability Discrimination.

effect over days ($F(2,12)=2.57$, $p=0.12$), nor the groups x days interaction ($F(2,12)=0.17$, $p=0.85$) was significant.

Finally, the responses to the preferred side during extinction, also calculated as percentages of the total daily responses, were analysed. The mean scores are shown in Figure 21 C, from which it can be seen that both groups showed very similar position habits on each of the three days. It is, perhaps, of interest to note that, compared with the last three days in reversal, the hippocampal pigeons made a small increase in their position responses in extinction, whereas the normal pigeons made a relatively larger decrease in position reponses. As expected, an analysis of variance carried out on the position preference scores in extinction showed that neither main effect, nor the interaction, was significant (all $F's<1$, all $p's>0.8$).

Discussion

During most of acquisition the normal pigeons responded to the majority rewarded colour on approximately 70% of trials, that is, they showed matching behaviour, whereas the hippocampal pigeons showed a general increase in their choice of the majority colour. However, although they showed a significant trend towards maximising over the 20 days, the differences between the two groups in acquisition, in fact, are not particularly large. However, the differences between the groups in the reversal stage of the experiment were much more pronounced. The hippocampal group showed a greater trend towards maximising, attaining a final score of 96.25% majority colour responses. On the other hand, the normal pigeons were retarded in reversal, responding to the majority stimulus on no more than about 60% of trials over days 5-20. It is clear, therefore, that the hippocampal pigeons performed more efficiently than the normal pigeons in both stages of the probability task, and particularly in the reversal stage.

The numbers of position responses made in the acquisition stage by the two groups were very similar, and over much of acquisition were maintained at a fairly constant

level, although the hippocampal pigeons reached this level somewhat earlier than did the normal pigeons. In fact, the hippocampal birds made marginally more position responses (mean, 64.2%) than the normal birds (mean, 58.2%), but these differences were not significant. However, the analysis of the majority colour responses shows that, despite similar levels of position responding by both groups, the responses of the hippocampal pigeons were controlled more by the relevant cue than by the irrelevant cue, since they gradually increased their choice of the majority stimulus. On the other hand, the normal pigeons maintained both their position responses and their majority colour responses at roughly constant and fairly similar levels throughout acquisition.

At the beginning of reversal, for the first four days, the normal pigeons continued to respond to their preferred position at about the same rate as they had during most of acquisition. At the same time they were responding to the new majority colour at chance level. (As a result of the extinction sessions following acquisition training, in which the two groups of pigeons were extinguished to approximately equal choice of the red and green keys, both groups began the reversal stage of the experiment by responding to the majority stimulus at chance level.) They then increased their position responding from just below 60% to 75%, which they maintained for the remaining 12 days of reversal training, and increased their majority colour responses from 50% on day 4 to 60% on day 5, continuing with this level of response to the relevant cue until the end of reversal. In contrast, the hippocampal pigeons immediately adopted a fairly strong position habit at the beginning of reversal, responding to their preferred position on about 75% of trials for the first six days, and then progressively decreased their position responding until, at the end of reversal, it was at chance level. Throughout reversal, however, as noted above, there was a concomitant increase in their choice of the new majority colour, from little over 50% on day 1 to just under 100% on day 20. Once again, therefore, these results show that, while the responding of the hippocampal pigeons was clearly

under the control of the majority colour (the relevant cue), the responses of the normal pigeons were controlled mainly by position. As noted in the Introduction (p.97), Mackintosh and Holgate (1968) showed that normal rats were also retarded on the reversal of a 75 : 25 brightness probability discrimination by the development of position habits.

Although the hippocampal pigeons made more responses than the normal pigeons on each of the three extinction sessions following the acquisition stage, none of these differences was found to be significant. Furthermore, in the three extinction sessions that were given after the completion of reversal training, the normal pigeons made rather more responses on the first day, and marginally more responses altogether in the three days, than the hippocampal pigeons, although neither difference was significant. These results therefore show that hippocampal lesioned pigeons, like hippocampal lesioned rats (e.g., Stevens, 1973c; see also the Introduction to the present experiment), need not be impaired on tasks involving response inhibition, and consequently are not consistent with the hypothesis that hippocampal damage results in an impaired ability to withhold responses.

Douglas and Pribram (1966) found that hippocampal lesioned monkeys performed less efficiently than normal monkeys on both the acquisition and reversal of a 70 : 30 visual probability discrimination, and their explanation was that hippocampal animals were less able to ignore a stimulus which provided occasional rewards, in order to respond consistently to a more frequently rewarded stimulus. Interestingly, Douglas and Pribram did not give their monkeys correction trials, and thus the animals were not actually forced to attend to and to respond to the minority rewarded stimulus. Had they done so, according to their model the hippocampal monkeys should have been even more impaired. Since the lesioned pigeons in the present experiment performed more efficiently than the normal pigeons in both stages of the experiment, it follows that the present results do not demonstrate impaired attention as proposed by Douglas and Pribram.

However, an alternative hypothesis has been proposed by Kimble (Kimble, 1968; Silveira and Kimble, 1968; Kimble and Kimble, 1970) in which it is suggested that the hippocampus is involved in selective attention and the formation of hypotheses. Silveira and Kimble (1968) and Kimble and Kimble (1970) found that rats trained on the acquisition, reversal, or extinction of a simultaneous brightness discrimination made longer runs of particular types of responses compared with normal rats, and they referred to this as hypothesis behaviour, as Krechevsky (1932) had done earlier with specific reference to discrimination learning in normal rats. They therefore suggested that hippocampal lesioned animals are impaired in their ability to change their hypotheses and that this is due to a defective selective attentional system. Stevens (1973a) found that hippocampal rats trained on a 70 : 30 spatial probability discrimination, without guidance, performed more efficiently than normal rats, and he proposed that, since his results, like the present ones, did not support the Douglas and Pribram model, the data could be better accommodated in terms of a mechanism in which the hippocampus was involved in the selection and rejection of hypotheses. He argued that, since there is evidence that rats initially respond to position in a simultaneous discrimination (Turner, 1968 - cited in Stevens, 1973a), they should begin by responding to position in a spatial probability task. But whereas normal rats are affected by the inconsistent reinforcement they receive, and as a result try out other hypotheses before finally returning to position responding, the hippocampal rats are slow to change their hypotheses and consequently continue their position responding and therefore perform more efficiently on the spatial probability task. Nevertheless, the finding of superior performance in the hippocampal rats, despite receiving 70% reinforcement on their nonpreferred side, is slightly puzzling in the light of reports that hippocampal animals tend to be impaired in the acquisition of a (100 : 0) spatial discrimination if the correct side is their nonpreferred side (see O'Keefe and Nadel, 1978). However, this could be resolved readily by assuming, as suggested in the Introduction, that the minimal pretraining the rats received was insufficient to allow

position preferences to be determined reliably, and that the rats were therefore not necessarily trained against their position preference. Indeed, in comparison with the five position preference trials that Stevens used, Samuels (1972) gave her rats five free trials and five forced trials a day for five days in order to ensure equal experience of both areas of the maze and to determine each rat's position preference.

It was noted in the Introduction that there is evidence to suggest that, for birds, visual cues are more salient than spatial cues. It might therefore be expected that, in a simultaneous visual discrimination, birds would initially respond to the visual cues, and there is some evidence to support this. Jones (1954) trained pigeons on an ambiguous-cue discrimination in which the stimulus dimensions were position, colour and form, and on test trials he found that four out of the six pigeons responded consistently to colour, while the other two responded to position. (This therefore suggests that the salience of visual and spatial cues for birds is relative rather than absolute, and that simple visual cues, such as colour, and perhaps brightness, are more salient than spatial cues, which in turn are more salient than pattern stimuli.) If Stevens' analysis is correct, then both normal and hippocampal lesioned pigeons, when trained on a colour probability discrimination, should initially respond to the visual cues, but inconsistent reward should cause the normal pigeons to try out other hypotheses, whereas the hippocampal pigeons would be expected to maintain their visual hypothesis and therefore would respond more efficiently. This is precisely what was found in the present experiment. However, Stevens (1973a), like Kimble, maintained that the fixated hypothesis behaviour of the hippocampal animals was due to impaired selective attentional processes, and Silveira and Kimble (1968) proposed that fixated attention to one stimulus dimension was associated with reduced attention to other cues; thus "once a hippocampal animal considers a stimulus important, attention to other stimuli is inhibited for a long time and little, positive or negative, is learned about these other stimuli" (p. 629). But the present

results do not support this proposal, since the hippocampal pigeons, throughout reversal training, despite correction trials, progressively increased their responses to the majority colour but maintained a fairly strong position habit for the first 600 trials. Although not as pronounced, a similar effect was also found during acquisition, in which the hippocampal pigeons gradually increased their responses to the majority colour while responding to their preferred position, on average, on 64% of trials, and over at least days 5 - 20 these scores were found to be reliably above chance. Thus, in both stages of the experiment, the hippocampal pigeons were obviously learning about the visual stimuli whilst responding to some extent to position. Related to this is the finding by Olton (1972a) that, when trained on a simultaneous pattern discrimination, hippocampal rats showed a clear discrimination, in terms of significantly different response latencies, between the positive and negative stimuli while still adopting a position habit. Therefore their attention was unimpaired, since they were able to gain information about the relevant cue while their choice behaviour was being controlled by the irrelevant cue. However, he also found that the hippocampal animals continued to respond to their preferred side significantly longer than the normal animals. Olton therefore argued that hippocampal animals were capable of normal levels of response suppression, but were impaired in their ability to shift their responding to the appropriate stimulus. This, of course, is very much like the proposal, referred to above, which suggests that hippocampal animals have abnormal difficulty in changing their hypotheses, but, unlike the mechanism proposed by Stevens and Kimble, does not involve impaired attentional processes.

A similar, although much more elaborated, explanation is offered by the theory of O'Keefe and Nadel (1978), in which the hippocampus is seen to be involved in spatial memory (see pp. 6-8). It would seem that the present data may be explained in terms of this theory as follows: Since the normal pigeons were able to adopt a place hypothesis

they soon began to respond appropriately to colour, because of its particular salience, but due to the flexibility of behaviour afforded by the use of place hypotheses, and their lapses of attention, they also began to reward-follow on position, just as chicks did in a brightness probability task (Mackintosh, 1969). Despite the three extinction sessions following acquisition, the normal pigeons began reversal with the same proportion of position responses as they had made during much of acquisition, but their learning of the reversal was impaired subsequently by the adoption of a position habit, as Mackintosh and Holgate (1968) had found in rats that were trained on the reversal of a brightness probability discrimination. Again, this would be possible because of the proposed nature of place hypotheses and, according to Sutherland and Mackintosh (1971), was due to inadequate attention to the relevant cue.

The hippocampal pigeons, on the other hand, while making approximately the same proportion of position responses as the normal pigeons, were not reward-following on position to the same extent, but apparently were adopting orientation and guidance hypotheses towards the majority colour, again, probably because of the salience of colour for birds. Because of the supposed inherent inflexibility and persistence of this non-hippocampal system, the hippocampal pigeons were able to gradually increase their choice of the majority stimulus. In reversal this behavioural rigidity was even more marked. At the beginning the hippocampal pigeons immediately adopted an inappropriate position habit, but at the same time were increasing their responses to the new majority colour, due to the use of guidance and orientation hypotheses only, and presumably, therefore, they were not responding consistently to position, i.e., reward-following on position, in the way that the normal pigeons were. Further evidence that the hippocampal pigeons would readily adopt an inappropriate position hypothesis comes from a comparison of the position habits adopted at the end of one phase of the experiment and the beginning of another. These are shown for the two

groups in the following table (Table 2). From this it is fairly clear that, whenever

	HIPPOCAMPALS	NORMALS
Last day of acquisition to first extinction session	57% - 66%	57% - 58%
Last extinction session to first day of reversal	55% - 75%	50% - 59%
Last day of reversal to first extinction session	54% - 64%	80% - 64%

Table 2 Comparison of position responses on the last day of one phase and the first day of the next phase for the two groups of pigeons.

the conditions of the task, changed, the hippocampal pigeons were much more prone to adopt a position hypothesis than were the normal pigeons, and O'Keefe and Nadel (p. 281) have commented on the ease with which hippocampal animals adopt persistent habits. However, in the present experiment, an important factor was undoubtedly the salience of the colour cues for the pigeons, which, in O'Keefe and Nadel's terms (p. 92), allowed a colour hypothesis to overshadow a position hypothesis.

It is proposed, therefore, that the present results support the proposal that hippocampal lesions in pigeons produce similar behavioural effects to those following hippocampal lesions in mammals. Furthermore, these results confirm other findings from experiments on mammals that hippocampal lesions do not cause a loss of response inhibition or impair attentional processes. However, they do support the hypothesis that the hippocampus is involved in place learning or spatial memory, and they appear to be consistent with the theory proposed by O'Keefe and Nadel (1978).

CHAPTER 4 Serial Reversals of a Spatial Discrimination

Introduction

In the visual probability task it was found that pigeons with hippocampal lesions did not reward-follow on position to the same extent that normal pigeons did, and it was argued, therefore, that they were not responding to spatial cues in the same way that normal pigeons were. Since there was evidence to show that the lesioned pigeons were not suffering from impaired attentional processes, or from a response inhibition deficit, it was concluded that the behavioural changes produced by the hippocampal lesions could be explained most readily in terms of impaired spatial ability.

A task which clearly requires normal spatial ability for its efficient performance is the serial reversal of a spatial discrimination, and it is now well-established that mammals with hippocampal lesions are impaired in their ability to learn such a task. The earliest report of a hippocampal deficit in rats on a spatial reversal task was by Thompson and Langer (1963), in which they used an avoidance-learning paradigm. Other reports of deficits in rats, but using a more conventional, positive-reinforcement situation, came from Kimble and Kimble (1965), Niki (1966), Hirsh and Segal (1972), Nonneman, Kolb, and Voigt (1974), and others. Similar impairments have also been found in cats by Brown, Kaufmann, and Marco (1967, 1969) and Uretsky and McCleary (1969), and in monkeys by Mahut and Cordeau (1963), Mahut (1971), Jones and Mishkin (1972), and Mahut and Zola (1973).

It is of some interest that all of the experiments on rats have used a T- or a Y-maze, or a variation of it (e.g., Cohen, LaRoche, and Beharry, 1971, and Cohen and LaRoche, 1973, used a +maze, and Nonnemann et al, 1974 used a Grice box), and the experiments on cats and monkeys were carried out using a Wisconsin General Test Apparatus (WGTA). Woodruff and Isaacson (1972) suggested that, since hippocampal lesioned rats often develop persistent position habits in a T-maze, discrimination tasks

should be carried out in an apparatus which minimises position responding, since this represents a confounding factor. Rather peculiarly, however, they then suggested that such an apparatus is an operant chamber, since "animals with hippocampal lesions do perform well in them and their performance is not confounded by position preferences" (p. 489). Thus, they trained hippocampal lesioned rats on a brightness discrimination in a two-lever operant chamber, and found that the lesioned animals were impaired, compared with normal rats, mainly because they showed a greater tendency to repeat inappropriate responses on the same lever, i.e., they adopted a position preference. In many respects the WGTA provides a situation which is very similar to that provided in an operant chamber, and thus various types of discrimination task are presented in a similar manner in either apparatus. Since, as noted above, both cats and monkeys with hippocampal lesions have been found to be impaired on spatial reversals when trained in a WGTA, it would therefore seem that hippocampal animals also ought to be impaired on a spatial reversals task presented in an operant chamber.

The reversal of a spatial discrimination is a rather simple task, and performance on it has been measured invariably in terms of response choice. On this basis there are only two ways in which an animal with hippocampal lesions may be impaired. Either the animal continues to respond to the previously correct position, or it fails to respond consistently to either position, for much longer than a normal animal does. The former effect, which has been found to occur in hippocampal rats (Kimble and Kimble, 1965) has been taken as further evidence that these animals are perseverative in their behaviour due to their inability to inhibit responses (Douglas, 1967; Kimble, 1968, 1969; Altman et al, 1973). Alternatively, Olton (1972a) has suggested that hippocampal rats do not have particular difficulty in suppressing responses, but instead are impaired in their ability to shift their responses to the other cue. However, such a finding also supports the spatial memory model of O'Keefe and Nadel (1978). They

propose that the hippocampal animal, because it is no longer able to use place hypotheses, has to rely on guidance and orientation hypotheses, which lack flexibility and consequently give rise to particularly persistent patterns of behaviour. Thus, evidence of continued responding to the previously rewarded position does not allow a distinction to be made between the response-inhibition, the response-shift, and the spatial memory hypotheses of hippocampal function. However, there is evidence from studies of non-spatial reversal learning which shows that the hippocampal deficit on these tasks is not due to perseverative or persistent responding to the previously correct stimulus, but is due to the prolonged maintenance of a position habit (Isaacson, Nonneman, and Schmaltz, 1968; Silveira and Kimble, 1968; Olton, 1972a), and Mahut (1971) has argued that, when perseveration of responses occurs, it "may be the symptom rather than the cause of the observed impairment in performance" (p. 422). Furthermore, Mahut also pointed out that "most of the tasks on which consistent deficits (and perseverative errors) are found in animals with hippocampal ablations appear to share an important spatial aspect" (p.422), an observation that has also been made by Samuels (1972).

On the other hand, if the hippocampal deficit were due to inconsistent responding to either position, this would support neither the response-inhibition nor the response-shift hypotheses, but instead would suggest that the animals were unable to respond reliably to spatial cues, and therefore would support the spatial memory model. Unfortunately, in the majority of cases, hippocampal deficits on a spatial reversal task have been reported only in terms of trials and errors to criterion, without presenting any further evidence concerning the nature of the errors. However, in those few studies in which more detailed results have been presented (e.g., Niki, 1966; Hirsh and Segal, 1972), it appears that the hippocampal animals (rats in each case) were impaired partly because they had difficulty in giving up responding to the former correct position,

and partly because, once they had managed to abandon the previous position habit, they had difficulty in responding consistently to the correct position. Clearly this supports the spatial memory model of hippocampal function.

The present experiment was designed, therefore, with several specific points in mind. The primary purpose was to attempt to discover whether hippocampal pigeons, like hippocampal mammals, are impaired on a serial position reversal task. In the event of such a deficit, detailed analyses of the individual response data were planned in order to be able to specify the nature of the deficit. The finding that the hippocampal pigeons had difficulty both in giving up the previous position habit, and in responding consistently to the correct position, would provide evidence to support the hypothesis that the avian hippocampus is involved in spatial ability. This would therefore also support the proposal, made previously, that the performance of the hippocampal pigeons in the probability task could be explained best in terms of impaired spatial ability. The task was presented in an operant chamber, partly because pigeons can be trained much more readily in this apparatus than they can in a maze or a discrimination box, and partly because, as argued above, an operant chamber and a WGTA, in which hippocampal deficits on a spatial reversal task have been found, allow a discrimination problem to be presented in a similar manner. Finally, on the completion of reversal training the pigeons were given an extinction session in order to be able to compare the extinction behaviour of normal and hippocampal pigeons following spatial discrimination training in an operant chamber, and also in an attempt to confirm the finding in the previous experiment that the hippocampal pigeons did not show an extinction deficit.

Method

Subjects

Twelve pigeons were used, all of which had been trained briefly in a previous

experiment (the acquisition of a colour discrimination). They were maintained at 80% of their ad lib bodyweights for the duration of the experiment and water was freely available in their home cages. Six of the pigeons had been given bilateral hippocampal lesions, and the remaining six were either sham-operated or unoperated controls.

Apparatus

A two-key Campden Instruments operant chamber was used in which both keys could be lit with white light.

Procedure

Surgery

Details of the surgical procedures used are described in full in Chapter 2.

Pretraining

The pigeons were pretrained in two stages. In the first stage both keys were lit and a single keypeck response to either key switched off the keylights and presented food reinforcement for 3 secs, followed by a 2 secs ITI during which the keylights remained off. The houselight was on throughout and the subjects were each given 25 trials. This stage was used to determine each pigeon's position preference. In fact, all animals responded to the left-hand key on most or all of the trials. Consequently, in the second stage, which took place on the following day, the animals were presented with a forced-choice situation in which only the preferred (in all cases the left) key was lit. A single response extinguished the keylight and was reinforced as before, followed by a 2 secs ITI. Again, the houselight remained on continuously and each pigeon was given a further 25 trials. In both stages the unlit keys were inoperable, but responses to them could be recorded.

Training

On the day following the completion of the 50 pretraining trials serial position

reversal training began. At the start of each trial the houselight was on and both keys were lit with white light. On the first reversal the key on the pigeon's nonpreferred side (in all cases, the right-hand key) was correct, and a single response to it turned off both keylights and was reinforced with 3 secs access to food. An incorrect response also extinguished the keylights, but was followed instead by 3 secs TO during which the houselight was turned off. A 2 secs ITI followed either reinforcement or TO, during which the houselight was on but the keylights were off and inoperable, although responses on them could be recorded. At the end of the ITI the keylights came on again for the start of the next trial. All pigeons were given 50 trials a day and were run until they reached a criterion of 9 correct responses out of 10 on each of two consecutive blocks of 10 trials. On the following day the next reversal began, and training to criterion continued as before. This procedure was repeated until each pigeon had completed ten reversals. On reversals 1, 3, 5, 7, and 9 the right-hand (originally nonpreferred) key was correct, and on the remaining reversals the left-hand key was correct.

After completion of the last reversal, on the following day, each pigeon was given a single extinction session of at least 50 trials, or at least 10 mins duration. At the start of the session both keys were illuminated with white light and a single response to either key extinguished both keylights for 5 secs. The houselight stayed on throughout the session, and once again the unlit keys were inoperable but responses on them were recorded.

Although electromechanical counters were used to record total numbers of correct and incorrect responses, the individual sequences of responses were recorded manually. In addition, all unlit key responses were recorded, and during extinction the time taken to complete 50 trials and the number of trials completed in 10 minutes were also recorded.

Results

Histology

Reconstructions of the lesions that were produced in the pigeons used in this experiment and in the following one (Chapter 5) are presented in Figure 22. Unfortunately the brain of one of the pigeons (No. 21) was lost during processing, but there are no reasons to believe that the hippocampal lesions in the other five pigeons are not representative of the lesion that was made in the brain of pigeon No. 21. The first impression is that the bilateral hippocampal lesion in these pigeons are comparable to those that were produced in the pigeons in the first experiment (Chapter 3). Here the lesions in the hippocampal group extend from A8.50 to A3.50, but again, in each pigeon the lesions are approximately 4 mm long. In all five pigeons the main lesion is in the hippocampus pars dorsalis, with relatively smaller amounts of damage occurring in the hippocampus and also in the parahippocampal area (APH). In two pigeons (Nos. 29 and 45) a small amount of damage can also be seen to have occurred in the accessory hyperstriatum (HA) at level A8.50, but in comparison with the extent of the hippocampal damage in these animals, and with the extent of the hyperstriatal damage that has commonly been produced in studies of the effects of hyperstriatal lesions (e.g., Macphail, 1975a), the amount of hyperstriatal damage produced here would appear to be minimal. In most of these pigeons minimal amounts of damage also occurred in the ventral hyperstriatum (HV), and in one pigeon (No. 29) minor invasion of the neostriatum (N) occurred at level A3.50.

Pretraining

Each pigeon's position responses in Stage 1 of pretraining are shown in Table 3, and it can be seen that the difference between the numbers of left key responses that were made by the two groups was minimal (Mann-Whitney $U=15.5$, $p>0.70$). The responses to the unlit keys in both stages of pretraining are shown in Table 4. In

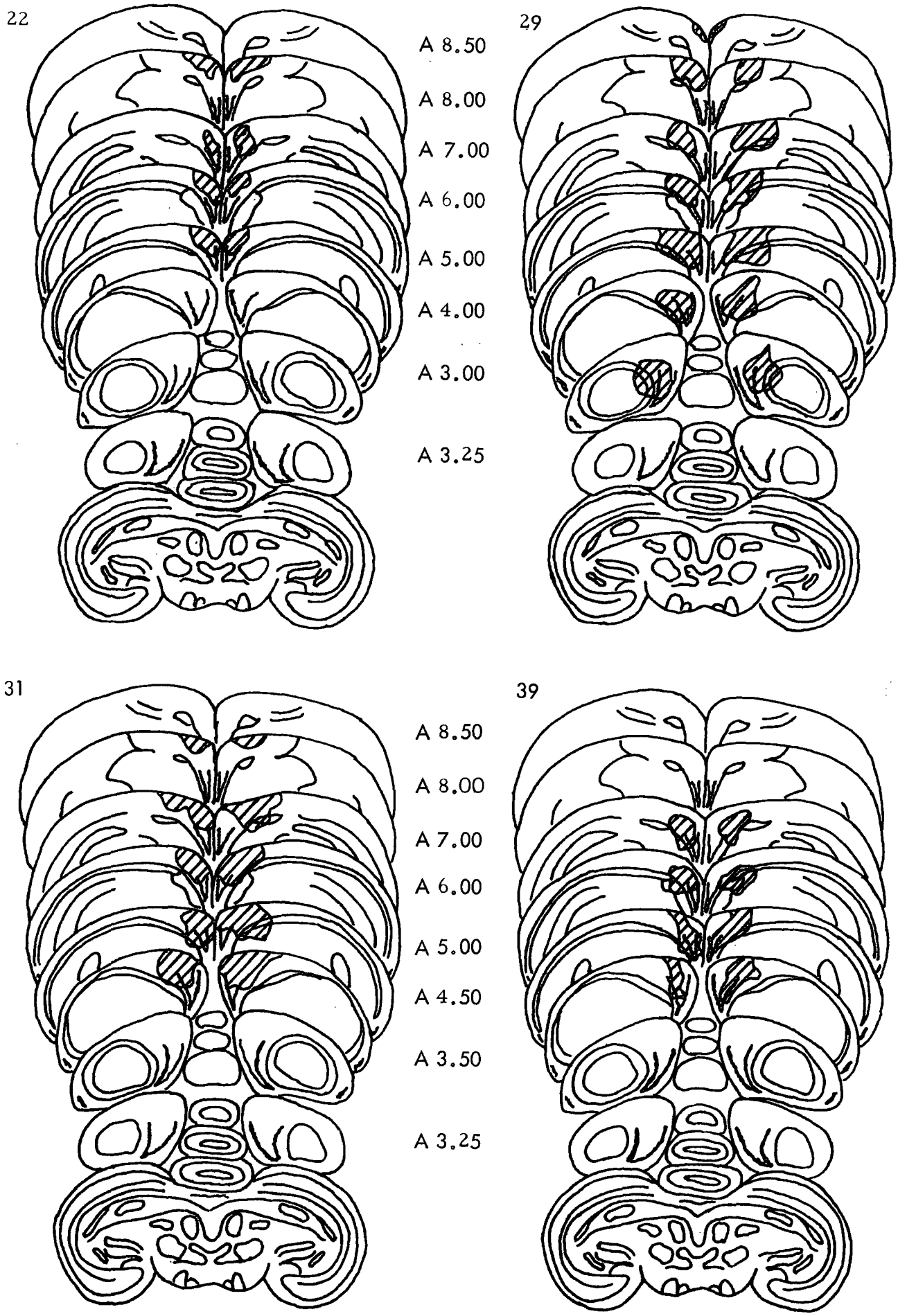


Figure 22. Reconstructions of the hippocampal lesions in five of the six experimental pigeons used in this and in the following experiment (see text). Stereotaxic coordinates correspond to those of Karten and Hodos (1967).

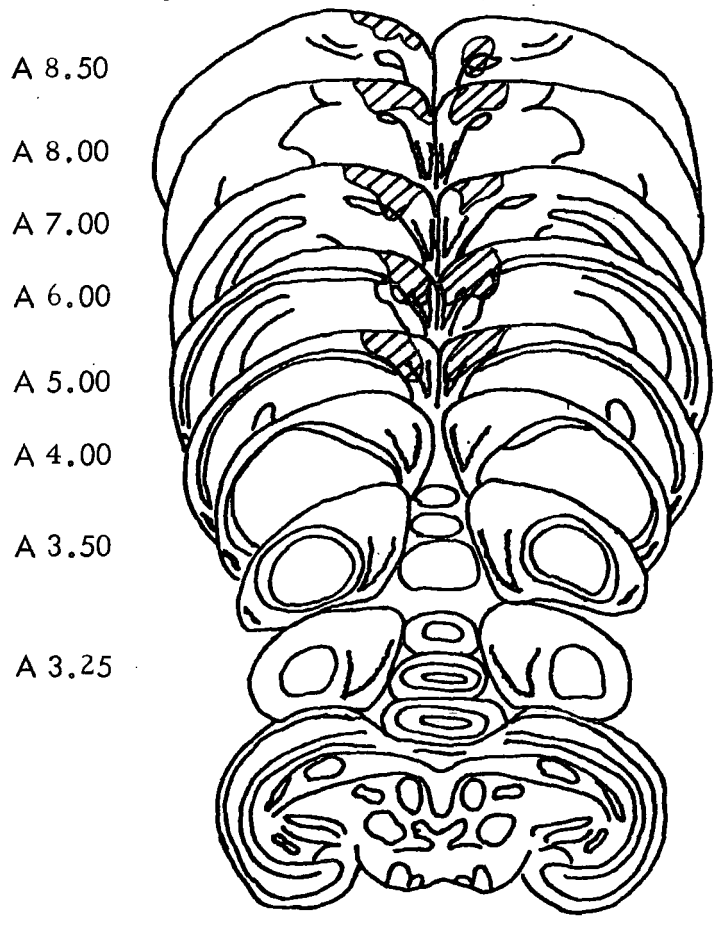


Figure 22 (contd.)

Table 3

Position responses in Stage 1 of pretraining

Normals

Subject	Left	Right
33	19	6
35	22	3
36	16	9
38	25	0
46	19	6
47	22	3
Means	20.5	4.5

Hippocampals

Subject	Left	Right
21	17	8
22	25	0
29	18	7
31	13	12
39	21	4
45	23	2
Means	19.5	5.5

Table 4

Responses to unlit keys in Stages 1 and 2 of pretraining

Normals

	Stage 1		Stage 2	
Subject	L	R	L	R
33	16	6	15	8
35	0	0	0	0
36	0	0	0	0
38	2	0	15	0
46	13	2	1	0
47	0	0	0	0
Total	31	8	31	8
Means	5.1	1.3	5.2	1.3

Hippocampals

	Stage 1		Stage 2	
Subject	L	R	L	R
21	80	27	15	0
22	13	8	2	0
29	5	2	15	0
31	86	67	58	22
39	3	0	0	0
45	26	0	13	0
Total	213	104	103	22
Means	35.5	17.3	17.2	3.1

Stage 1 the hippocampal pigeons made more unlit left key responses than the normal pigeons, but this difference was found to be only marginally significant (Mann-Whitney $U=5.5$, $0.064>p>0.042$). They also tended to make more unlit right key responses than the normal pigeons, but these scores were not significantly different (Mann-Whitney $U=9.5$, $p>0.18$). In Stage 2 neither of the differences between the two groups was significant, although again, the hippocampal pigeons tended to make more unlit left key responses than did the normal pigeons.

Thus, in pretraining all animals showed a marked initial preference for the left key, which was clearly not affected by the hippocampal lesions. However, the hippocampal group did show a much greater tendency than the normal group to respond to the unlit keys, although only the left key responses in Stage 1 were found to be significantly greater for the hippocampal pigeons, and then only marginally so.

Serial Reversal Training

The mean numbers of trials and errors to criterion, including the 20 trials of the criterion run, for each of the two groups over the ten reversals are presented in Figure 23, and analyses of variance showed that the hippocampal pigeons took significantly more trials ($F(1,10)=9.77$, $p=0.011$) and made significantly more errors ($F(1,10)=8.01$, $p=0.017$) than the normal pigeons. The performance of both groups over reversals is shown in terms of trials to criterion in Figure 24, and for errors to criterion in Figure 25. The analyses of variance confirmed that there was a significant reduction in both trials ($F(9,90)=6.82$, $p<0.00005$) and errors ($F(9,90)=10.56$, $p<0.00005$) over reversals, but that the groups x reversals interaction was not significant for trials ($F(9,90)=1.55$, $p=0.14$), although this interaction was significant for errors ($F(9,90)=2.31$, $p=0.022$), showing that the hippocampal pigeons reduced their errors over reversals to a greater extent than the normal pigeons did. Analysis of the simple main effects then revealed that the hippocampal pigeons made significantly

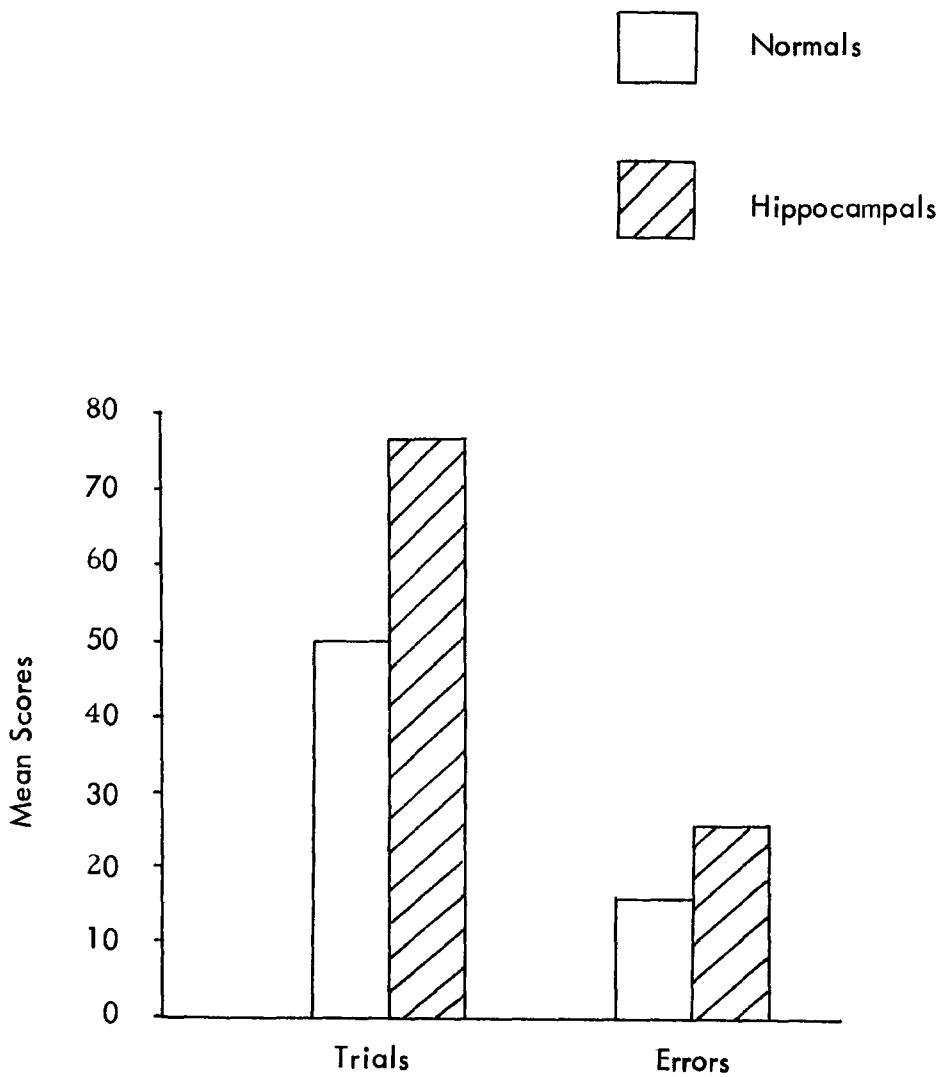


Figure 23. Mean Trials and Errors over the 10 Reversals

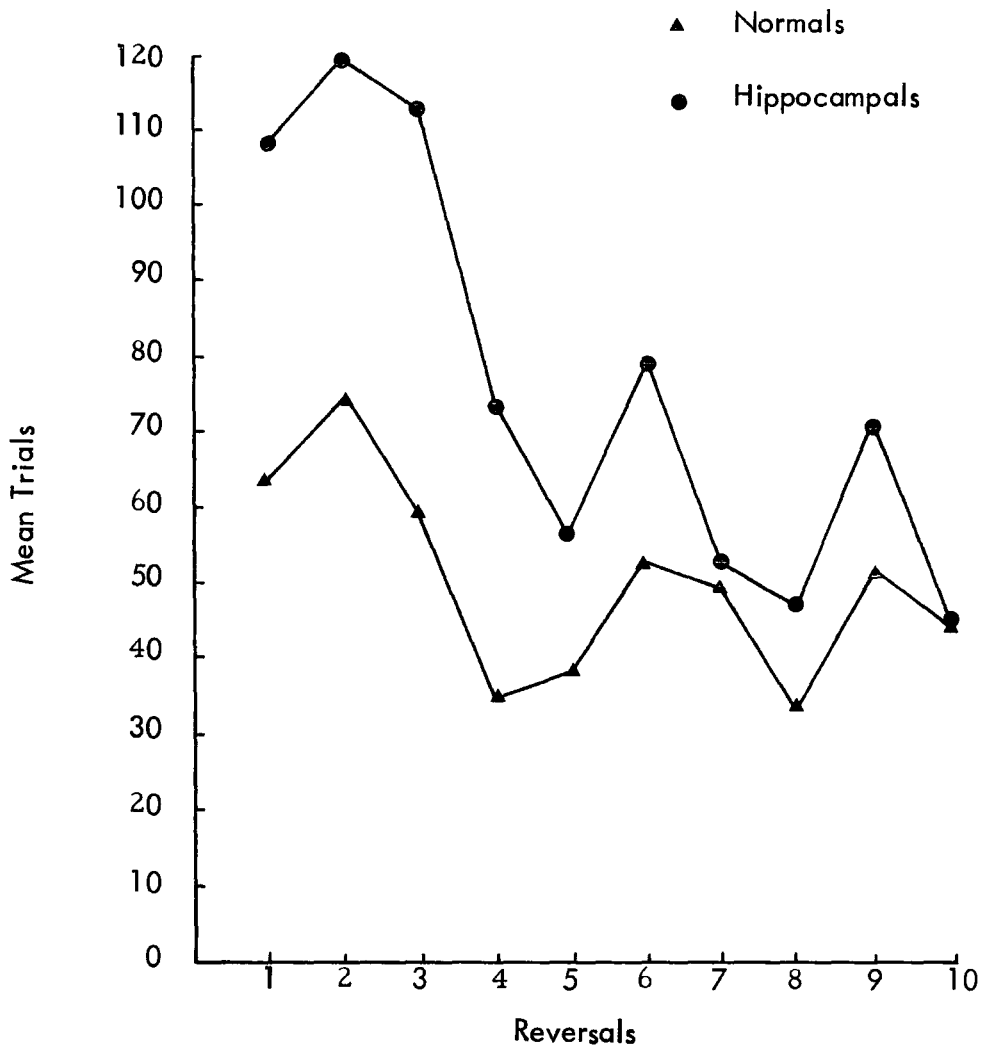


Figure 24. Mean trials to criterion on each of the ten reversals.

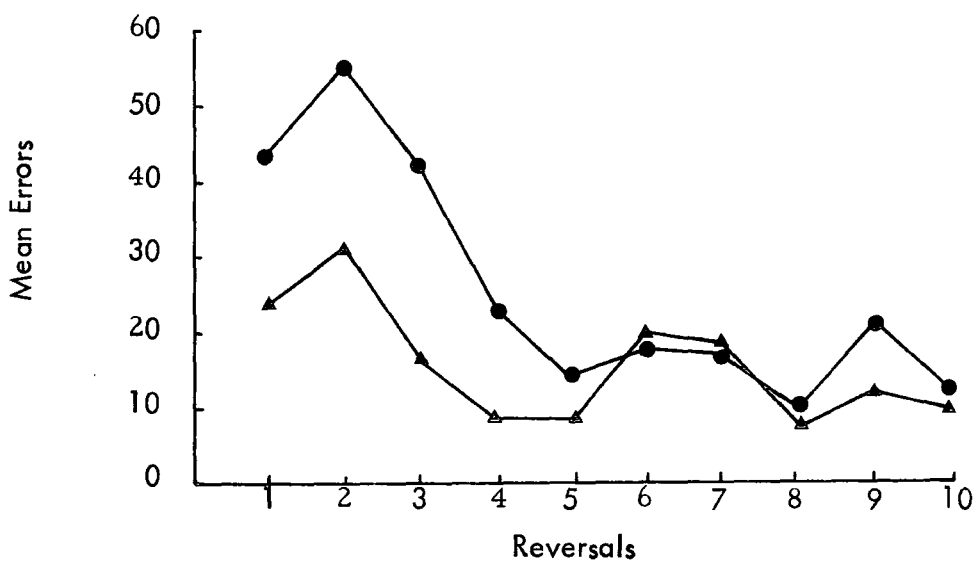


Figure 25. Mean errors to criterion on each of the ten reversals.

more errors than the normal pigeons only on reversals 1-4 ($F(1,10)>5.8$, $p<0.035$ in each case). These results, therefore, show that the hippocampal pigeons were impaired, compared with normal pigeons, when trained on serial spatial reversals in an operant chamber.

In order to analyse in more detail the effects of hippocampal lesions on this task, a number of additional measures were obtained from the raw data. Since the initial stage of reversal learning demands that animals learn not to respond to the previous positive stimulus, and since a number of investigators have found that hippocampal lesions in rats (e.g., Kimble and Kimble, 1965; Niki, 1966; Cohen et al, 1971) produced marked response perseveration in a position reversal task, the first score obtained from the data was for the number of errors to the first correct response. This provided a direct measure of the perseverative responses made by each animal at the beginning of each reversal. These scores, which are presented in Figure 26, were subjected to an analysis of variance, which showed that the hippocampal pigeons did not tend to make more responses to the former positive stimulus at beginning of each reversal ($F(1,10)=0.14$, $p=0.71$). Together, the two groups showed a significant reduction in errors to first correct response over reversals ($F(9,90)=2.28$, $p=0.024$), but the groups x reversals interaction was not significant ($F(9,90)=1.36$, $p=0.22$).

The other measures that were obtained from the original data, and which are the same as those used by Macphail (1972) in his analysis of serial position reversal learning in normal pigeons, were 1) the numbers of correct responses made in each reversal, and 2) the numbers of errors in each reversal excluding those which preceded the first correct response. These are summarised in Figures 27 and 28 respectively. It can be seen from Figure 27 that, apart from the final reversal, the hippocampal subjects consistently made more correct responses per reversal than the normal pigeons. Analysis of variance showed that this effect was significant ($F(1,10)=10.52$, $p=0.009$), and

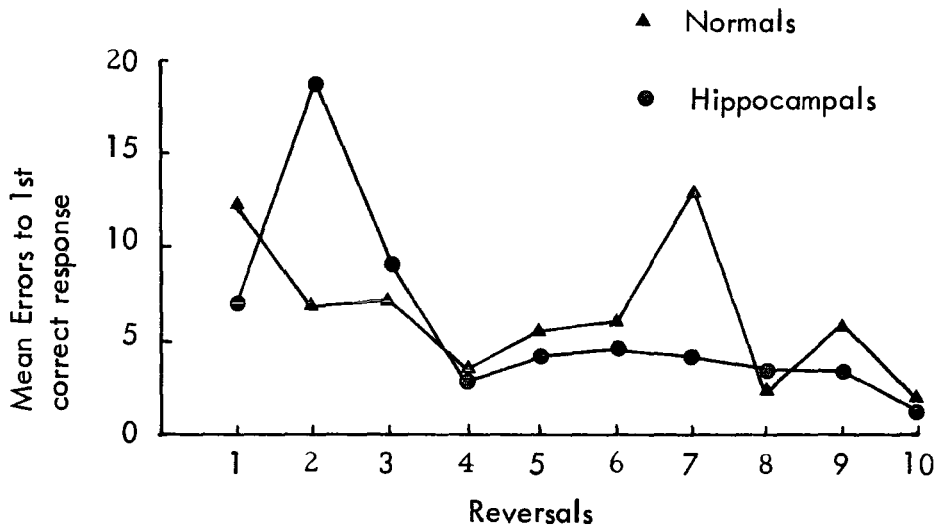


Figure 26. Mean errors to the first correct response made by the two groups of pigeons on each of the ten reversals.

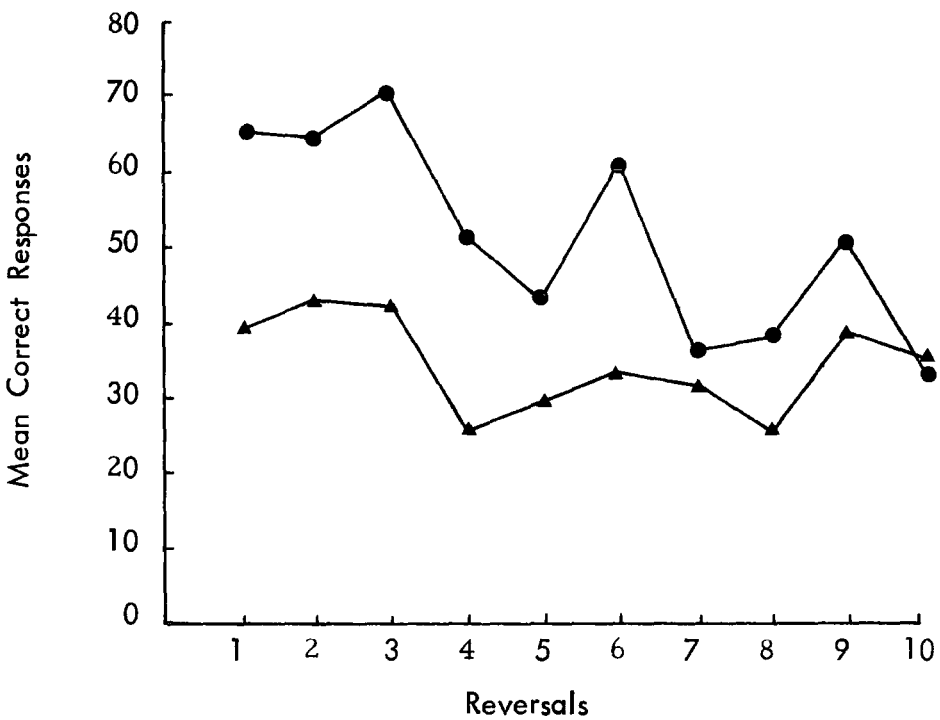


Figure 27. Mean numbers of correct responses made on each of the ten reversals.

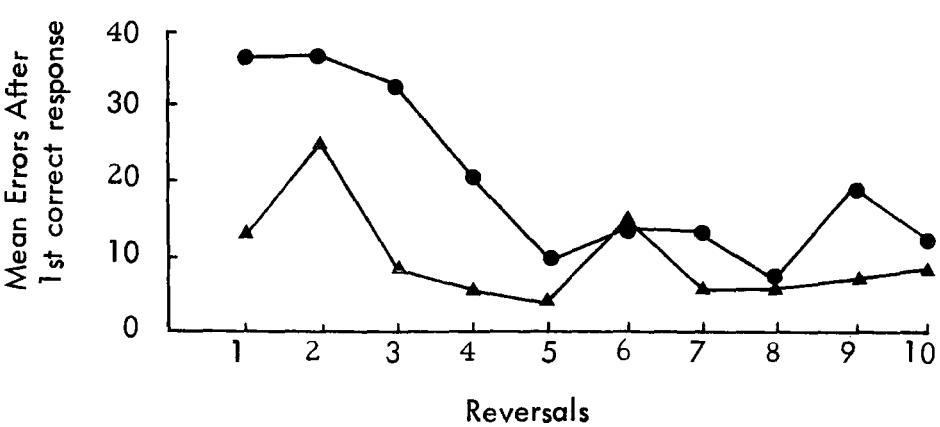


Figure 28. Mean numbers of errors made by each group following their first correct response on each of the ten reversals.

that there was a significant reduction in the numbers of correct responses over reversals ($F(9,90)=2.93$, $p=0.004$), but the interaction between the two groups over reversals was not significant ($F(9,90)=0.97$, $p=0.47$). The hippocampal pigeons also made consistently more errors after the first correct response, compared with the normal pigeons, on all reversals except reversal 6, and the analysis of variance confirmed that this effect was significant ($F(1,10)=7.90$, $p=0.018$). Also, there was a significant reduction in these errors over reversals ($F(9,90)=7.13$, $p<0.00005$), and the interaction between the two groups over days was significant ($F(9,90)=2.15$, $p=0.033$). An analysis of the simple main effects then showed that the hippocampal pigeons made significantly more errors after the first correct response than the normal pigeons only on reversals 1, 3, and 4 ($F=13.07$, $p=0.01$; $F=8.69$, $p<0.025$; $F=6.65$, $p<0.05$ respectively, with 1 and 10 df).

Thus, the impaired serial reversal performance of the hippocampal group was due, not to exaggerated perseverative responding to the previously correct position, but instead to the increased numbers of errors that they made after making their first correct response. However, they also made more correct responses than the normal pigeons on each reversal, except the final one, and when the total errors scores were calculated as percentages of the trials on each reversal, it was found that the proportions of errors made by each group over all ten reversals were very similar (means: normal group, 29.9%; hippocampal group, 31.3%). The percentages of errors on each reversal are presented for the two groups in Table 5. This suggests, therefore, that the hippocampal pigeons were not maintaining a particular response for as long as the normal pigeons, but instead were switching responses more frequently, the frequency depending on the lengths of the response sequences involved.

Figures 29 and 30 summarise the results of an analysis of the runs of correct responses and of errors made by each group, the mean frequencies having been obtained

Table 5

Percent errors

Reversal	Normals	Hippocampals
1	37.9	39.9
2	41.7	46.1
3	27.4	37.3
4	24.9	37.7
5	23.0	25.0
6	37.9	22.9
7	36.4	32.8
8	23.1	20.3
9	24.6	29.7
10	22.2	27.3
Means	29.9	31.3

by combining the data from all ten reversals in each case. From these graphs it can be seen that the hippocampal pigeons made considerably more short length runs, particularly runs of 1 and 2 correct responses and errors. Although the differences do not appear to be as marked, the hippocampal group made approximately twice as many runs of 3, 4, and 5 responses, both correct and incorrect, as the normal group. However, run-lengths of 6 to 11+ responses appear to be very similar between the two groups. In order to indicate the reliability of these differences, standard deviations of the means have also been plotted in the figures, and from these it can be seen that the hippocampal pigeons made reliably more runs of 1 and 5 correct responses, and runs of 1 and 2 errors, than the normal pigeons, although the overlap in the standard deviations of the two groups is minimal for runs of 2 correct responses and for runs of 3 errors. This marked increase mainly in the frequency of runs of 1 and 2 correct and incorrect responses by the hippocampal group must mean that, compared with the normal group, they switched their responses between the positive and negative keys more frequently. However, since the hippocampal pigeons took more trials, making both more correct responses and more errors, than the normal pigeons, they clearly had more opportunities to make more short-length runs of responses. (But it can also be argued, of course, that it was because they made more short-length runs of correct and incorrect responses that they made more responses overall, and therefore took more trials to reach criterion on each reversal than the normal pigeons.)

In order to equate the scores of the two groups for opportunity to make particular sequences of responses, the run-length frequencies of correct responses and of errors were calculated as percentages of the total responses that each group made over the ten reversals. These measures are summarised in Figures 31 and 32, and from these graphs it can be seen once again that the hippocampal pigeons made greater proportions of sequences of 1, 2, 3, 4, and 5 correct responses and errors than the normal pigeons,

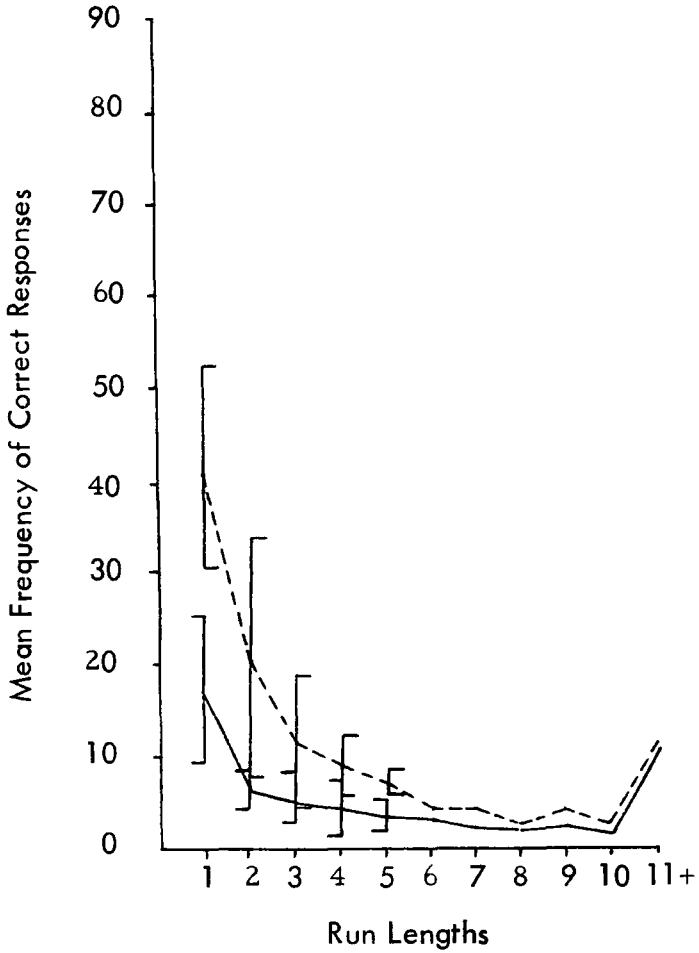


Figure 29. Frequencies of correct responses occurring in various run-lengths.

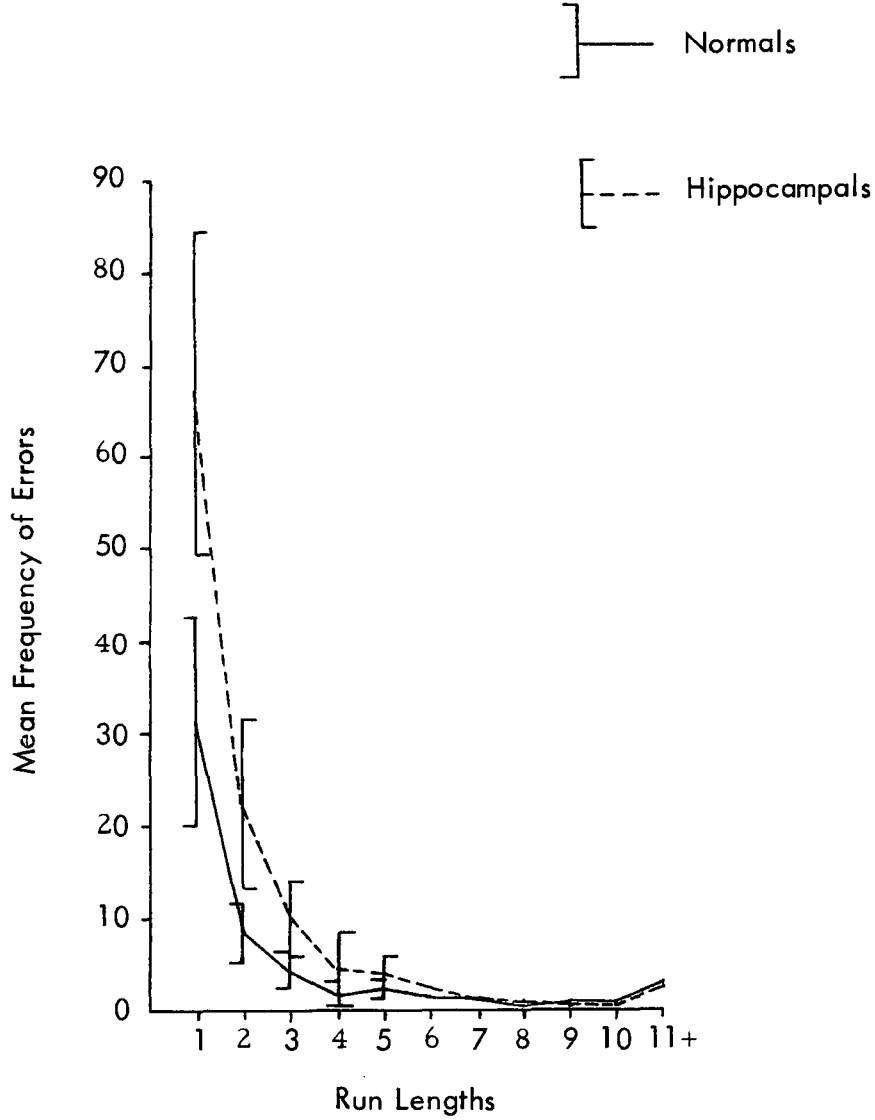


Figure 30. Frequencies of errors occurring in various run-lengths.

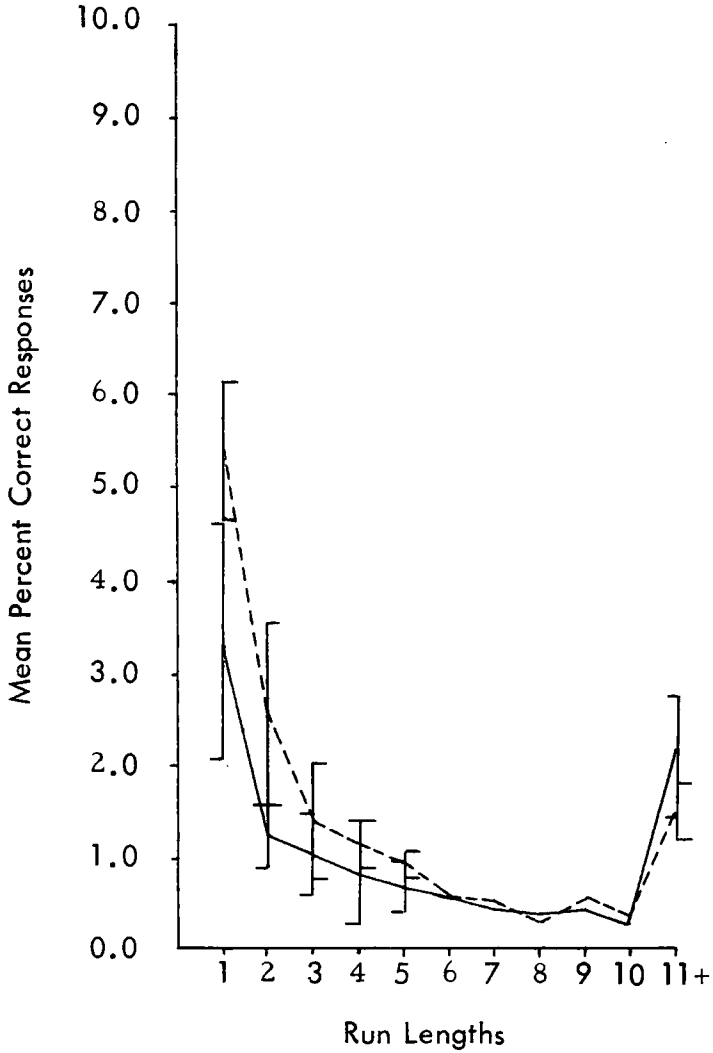


Figure 31. Percentages of correct responses occurring in various run-lengths.

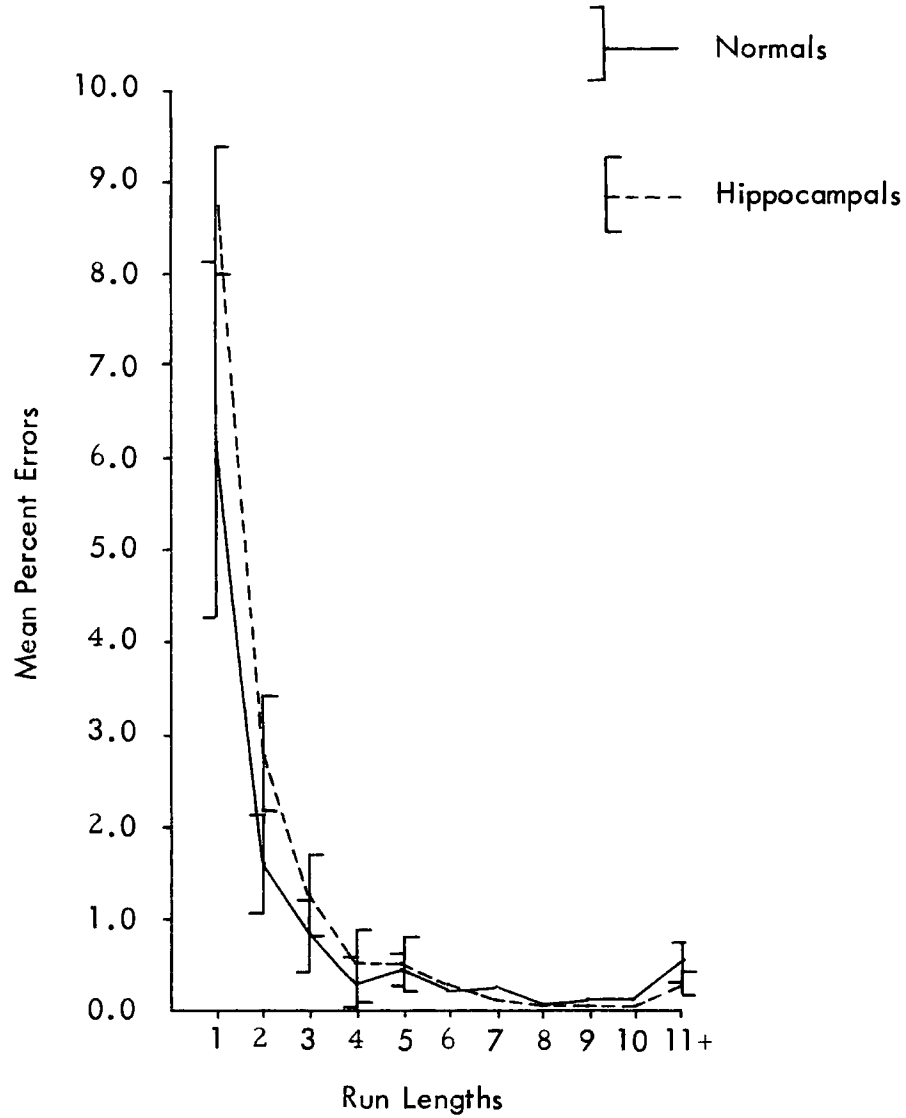


Figure 32. Percentages of errors occurring in various run-lengths.

while the normal pigeons tended to make greater proportions of sequences of 11+ correct responses and errors. However, as the standard deviations indicate, the scores of the hippocampal group were reliably higher only for runs of 1 and 2 correct responses and runs of 2 errors, although the overlap of the standard deviations of the scores for single errors was only very small. This analysis therefore suggests that, when the run-length frequencies were equated for opportunity, so that true comparisons between the groups could be made, the hippocampal pigeons made relatively more runs of 1 and 2 correct responses, and errors, compared with the normal pigeons.

Nevertheless, this analysis gave no indication of the order in which correct and incorrect responses occurred. It seemed, therefore, that a further analysis of the raw data was required, and a method that appeared to be both useful and appropriate to this experiment was that devised by Macphail (1976a) in order to analyse the individual response data from normal and hyperstriatal lesioned pigeons on a within-day serial position reversal task. The technique consisted of classifying trials as

a) perseverative errors, which included errors to the first correct response and any other error occurring in a sequence of two or more;

b) incomplete runs, which consisted of correct responses in sequences of two or more, excluding the criterion run;

c) alternation responses, consisting of sequences of two or more responses alternating between sides, excluding those trials which had already been classified as belonging to categories a) or b); and

d) unclassified responses, comprising single trials which intervened between sequences of trials already classified as perseverative errors or incomplete runs.

This technique was applied to the individual trials from the present experiment in the manner described by Macphail, with the exception that, in this experiment, each

daily 50 trial block was treated as a discrete unit for the purposes of this analysis, rather than counting each reversal as the discrete unit as Macphail did. Also, because the learning criterion in the present experiment was 9 out of 10 correct responses on two consecutive blocks of 10 trials, the criterion run that was excluded from the incomplete runs category consisted of the last 20 trials of each reversal. The data obtained from this analysis are presented for each of the four categories in Table 6. However, response categories a) and b) do not provide any information about mixed sequences of correct and incorrect responses, and anyway have already been presented in only a slightly different guise (the perseverative error scores as defined by Macphail are, in fact, very similar to the scores obtained for the errors to criterion on each reversal and which are presented in Figure 25; and the incomplete runs measure is very similar to the scores for correct responses in each reversal that are summarised in Figure 27). Consequently they will not be given further consideration here. On the other hand, by definition, the alternation responses category is a direct measure of the sequential nature of correct and incorrect responses, and therefore it provides information about response patterns. The unclassified responses category also provides some information about response patterns, since it consists of single responses, either correct or incorrect, that occur after runs of two or more incorrect or correct responses.

The mean numbers of alternation responses made by the two groups in each of the ten reversals are presented in Figure 33. An analysis of variance that was carried out on these scores showed that the hippocampal pigeons made significantly more alternation responses than the normal pigeons ($F(1,10)=19.11$, $p<0.002$), and that there was a significant reduction in these scores over reversals ($F(9,90)=2.46$, $p=0.015$), but that the interaction of lesion-treatment x reversals was not significant ($F(9,90)=1.18$, $p=0.32$). The unclassified responses are summarised in Figure 34 and were also subjected to an analysis of variance. This showed that the hippocampal pigeons made

Table 6

Mean trials, excluding criterion runs, in each of four categories of response on each of the ten reversals

NORMALS	Mean Trials										
Reversal	1	2	3	4	5	6	7	8	9	10	Means
a) Perseverative errors	19.5	25.7	11.3	7.3	6.2	16.3	16.0	4.7	10.5	7.7	12.5
b) Incomplete runs	17.7	21.3	21.8	6.5	8.8	12.7	11.8	5.0	19.2	14.3	13.9
c) Alternation	3.5	5.0	3.0	0.5	2.3	2.8	1.2	3.0	0.3	1.5	2.3
d) Unclassified	2.7	3.0	2.2	0.7	1.0	1.2	1.0	0.7	1.7	1.5	1.5

HIPPOCAMPALS	Mean Trials										
Reversal	1	2	3	4	5	6	7	8	9	10	Means
a) Perseverative errors	31.7	46.5	32.3	16.8	9.8	11.5	13.7	3.7	15.0	8.5	19.0
b) Incomplete runs	38.8	37.2	45.5	26.8	21.3	41.0	15.3	16.8	27.6	11.5	28.2
c) Alternation	10.3	9.2	10.2	6.2	3.2	3.8	2.8	2.7	6.0	3.7	5.8
d) Unclassified	7.3	7.2	5.3	5.1	2.3	3.7	1.5	1.8	3.0	1.3	3.9

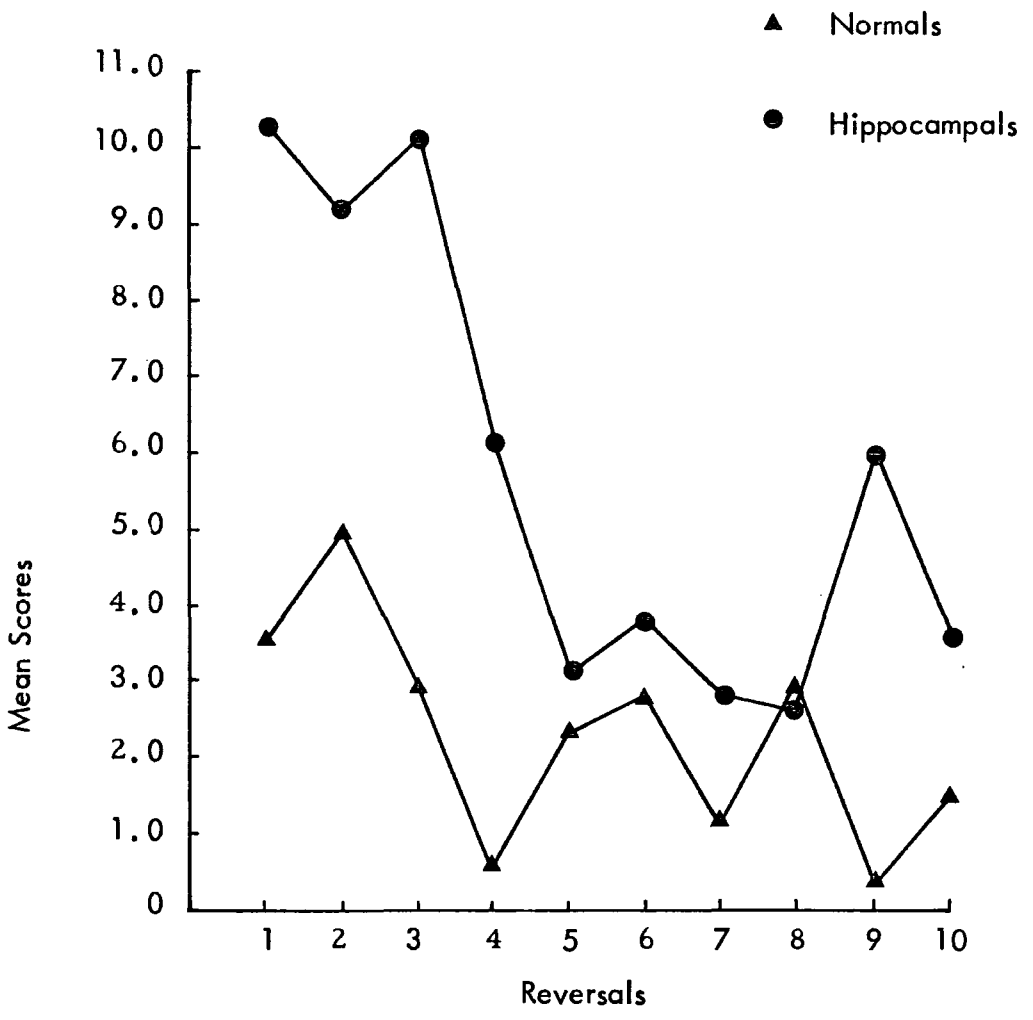


Figure 33. Mean numbers of alternation responses.

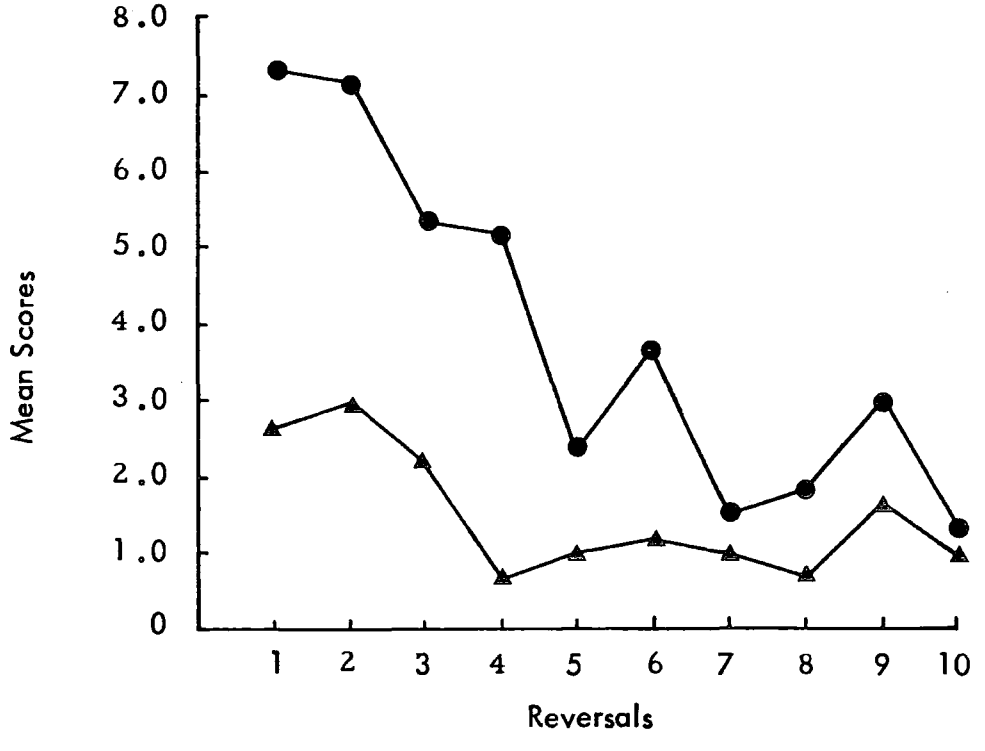


Figure 34. Mean numbers of unclassified responses.

significantly more single responses between runs of other responses than the normal pigeons ($F(1,10)=11.80$, $p=0.006$), and that there was a significant reduction in these responses over reversals ($F(9,90)=3.98$, $p=0.0003$), but that the groups x reversals interaction was not significant ($F(9,90)=1.41$, $p>0.20$). This analysis therefore confirms the finding from the analysis of runs of responses that the hippocampal pigeons made more single responses, either correct or incorrect, than the normal pigeons, and extends these findings by showing that the hippocampal group also made more alternating pairs of responses, in which a response on one key would be followed on the next trial by a response on the opposite key.

A further measure of persistent, inappropriate responding by the hippocampal pigeons was provided by an analysis of the extraneous responses that were made to the unlit keys. These were recorded separately for the correct and the incorrect keys and are presented for the two groups in Figure 35. A three-factor analysis of variance (lesion treatment x reversals x keys) was carried on these data and revealed that, overall, the hippocampal pigeons made significantly more unlit key responses than the normal pigeons ($F(1,10)=7.32$, $p=0.021$). There was also a significant reduction in these extraneous responses over reversals ($F(9,90)=3.80$, $p<0.001$), and together, both groups made significantly more responses to the unlit key on the correct side ($F(1,10)=19.65$, $p=0.001$). Significant interactions were also found between the two groups over reversals ($F(9,90)=3.05$, $p=0.003$), showing that the hippocampal group made a significantly greater reduction in their total unlit key responses over reversals compared with normal group, between groups and keys ($F(1,10)=5.58$, $p=0.038$), showing that there was a greater difference between the correct and incorrect unlit key responses for the hippocampal group than there was for the normal group, and between correct and incorrect keys over reversals ($F(9,90)=2.80$, $p=0.006$), confirming that there was a significantly greater reduction over reversals in responses to the unlit key

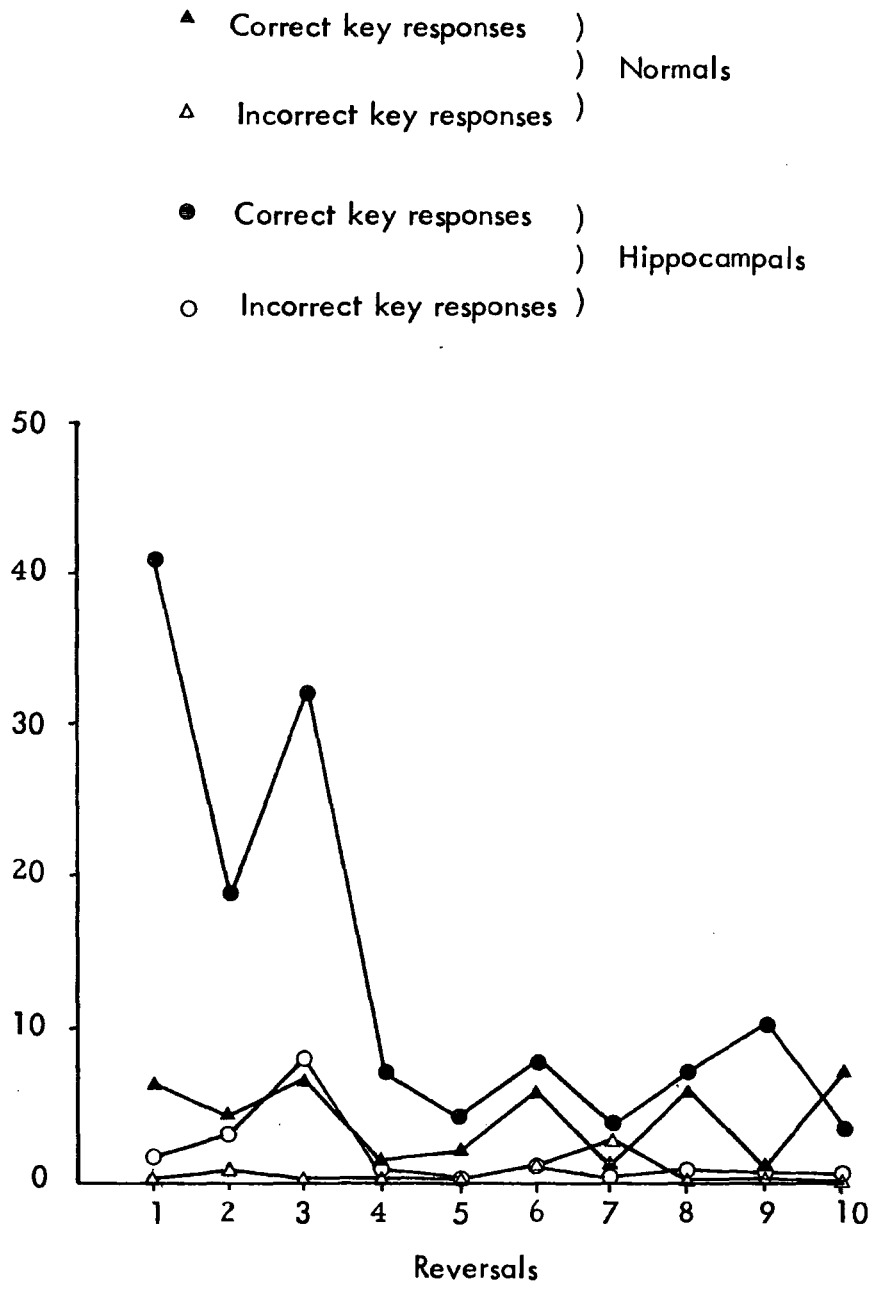


Figure 35. Mean responses to the unlit correct and incorrect keys on each of the ten reversals.

on the correct side than there was in the responses to the key on the incorrect side. It can be seen from the figure, however, that the major contribution to these effects came from the unlit correct key responses of the hippocampal group. This was confirmed by separate trend analyses carried out on the responses to the unlit keys on the correct and incorrect sides, which revealed that the linear component of the responses to the unlit correct key made by the hippocampal group was significant ($F(1,5) = 8.87$, $p < 0.05$), but that there was not a significant linear trend in the incorrect key responses of the hippocampal group ($F(1,5) = 5.15$, $p > 0.05$), or in the responses made by the normal group to either the correct key ($F(1,5) = 0.06$, $p > 0.25$) or the incorrect key ($F(1,5) = 0.02$, $p > 0.25$) when they were unlit.

In summary, the hippocampal pigeons made significantly more responses to the unlit key on the correct side, and showed a significantly greater reduction in these responses over reversals, compared with their responses to the unlit key on the uncorrect side, and compared with the responses of the normal group to either key when it was unlit. The numbers of responses made to the incorrect key by the hippocampal group, and to either key by the normal group, were relatively low and none of them showed any significant trend over reversals.

Finally, as might be expected from the overall results of this experiment, the hippocampal pigeons made significantly fewer reversals in 500 trials compared with the normal pigeons (means: hippocampal group, 5.5 reversals, normal group, 9.2 reversals; Mann-Whitney $U = 2.5$, $008 > p > 0.004$). These data for the individual subjects are presented in Table 7.

Extinction

The data collected during the single extinction session that followed the completion of the final reversal are summarised in Table 8, and it can be seen that the hippocampal pigeons took rather less time to complete 50 responses and tended to make more responses

Table 7

Number of reversals completed in 500 trials

Normals

Subjects	No. of reversals
33	10
35	8
36	10
38	10
46	7
47	10

Hippocampals

Subjects	No. of reversals
21	3
22	3
29	5
31	7
39	7
45	8

Table 8

Extinction data

Normals

Subject	Time for 50 responses (secs)	Number of responses in 10 minutes	Position responses	
			Left	Right
33	690	42	39	11
35	1009	27	35	15
36	454	63	30	33
38	520	57	35	22
46	>900*	37	27	10
47	520	57	29	28
Means	682.2	47.2	32.5	19.8

* stopped responding on trial 47 7 minutes after the start of extinction, and session terminated after 15 minutes.

Hippocampals

Subject	Time for 50 responses (secs)	Number of responses in 10 minutes	Position responses	
			Left	Right
21	670	47	21	29
22	469	62	22	40
29	570	53	32	21
31	532	56	26	30
39	437	69	39	30
45	523	50	38	12
Means	533.5	56.2	29.7	27.0

in a 10 min period than the normal pigeons. The two groups made very similar numbers of responses to the left-hand key during extinction (the side which was correct in the final reversal), but the hippocampal group showed a slightly greater tendency to respond to the right-hand key as well. However, none of these differences between the two groups was significant (in all cases, $U>10$, $p>0.12$, Mann-Whitney). Thus, although the hippocampal pigeons showed a greater tendency than the normal pigeons to respond in the absence of reinforcement, all of the measures used show that they were not impaired on this extinction task.

Discussion

The results of this experiment showed quite clearly that, in common with hippocampal-damaged rats, cats, and monkeys, pigeons with lesions restricted to the hippocampal complex were impaired on a serial position reversal task. Furthermore, they showed that the occurrence of such an effect does not depend on the spatial task being presented in a maze, a discrimination box, or a WGTA, but that it can also be found to occur in an operant chamber. However, the hippocampal pigeons in this experiment did not show an increased resistance to extinction following extended training in an operant task, showing that they were not suffering from a loss of response inhibition, and thereby confirming the results obtained in the previous experiment. A similar lack of an extinction deficit has also been reported in hippocampal rats trained in an operant chamber (Schmaltz and Isaacson, 1967; Nonneman et al, 1974). This finding suggested that the spatial reversal deficit was not due to an inability to withhold responses that were no longer rewarded, and support for this suggestion came from an analysis of the individual data, which showed that, compared with the normal pigeons, the hippocampal pigeons did not make more incorrect responses at the beginning of each reversal. Thus, the deficit was not due to exaggerated responding to the previously correct position, and therefore these results do not support the response-perseveration

hypothesis of the effects of hippocampal lesions (e.g., Kimble and Kimble, 1965; Uretsky and McCleary, 1969).

Further analysis of the data showed that the hippocampal pigeons tended to make more short runs of correct and incorrect responses, and that they alternated responses between the correct and the incorrect positions more than the normal pigeons did. They were impaired, therefore, because they had difficulty in making a sufficiently long run of responses to the rewarded position in order to reach the criterion of nine correct responses in a block of ten trials for two consecutive blocks. This demonstrates that they also were not impaired in their ability to shift responses, as had been suggested by Olton (1972a). Here it is important to note that Macphail (1975a,1976a) found that pigeons with hyperstriatal lesions were also impaired on the serial reversal of a position discrimination. However, from those results, and from the results of experiments on the effects of hyperstriatal lesions on the acquisition and reversal of non-spatial discriminations, Macphail argued that the hyperstriatal region is involved in a response-shift mechanism (Macphail, 1975a) or a response-inhibition mechanism (Macphail, 1971, 1976a, 1976b).

The finding, from the analysis of the individual data, that the hippocampal pigeons tended to respond sometimes randomly, and sometimes by repeating sequences of responses, either alternating sequences, or short runs, of correct or incorrect responses, is very similar to that reported by Olton, Walker, and Gage (1978). They found that rats with hippocampal lesions trained on a spatial memory task in an 8-arm radial maze tended to repeat sequences of arm-entries at above chance level, and also made a considerable number of errors that "appeared to happen haphazardly" (p. 305). This result suggested to Olton et al that the rats either were unable to make appropriate decisions about the places they had been to, or were no longer able to use extra-maze cues and hence were impaired in their ability to learn about places, an interpretation

favoured by O'Keefe and Nadel (1978). In either case, however, Olton et al argued that their results supported the hypothesis that the hippocampus plays a major role in the processing of spatial information.

Since the hippocampal pigeons had difficulty in responding consistently to the correct position, showed a similar pattern of errors to those reported by Olton et al, and were clearly not impaired in their ability to withhold or shift responses, it is proposed here that these results again support the hypothesis that, like the mammalian hippocampus, the avian hippocampus plays an important role in spatial ability. In their spatial memory model of hippocampal function, O'Keefe and Nadel propose that normal animals will initially use place hypotheses to solve a spatial reversal problem, but with continued training will shift towards using orientation hypotheses, which give rise to persistent response patterns. Because of their inability to use place hypotheses, hippocampal animals are forced to use guidance and orientation hypotheses from the very beginning. However, besides being prone to persistence, guidance and orientation hypotheses are also liable to interference effects, and because hippocampal animals are not able to respond appropriately to spatial cues, i.e., they have difficulty in knowing where to respond, they are much more likely to make inappropriate responses in a spatial task, and to repeat them. On the other hand, an extinction task in an operant chamber makes very little use of place hypotheses, but instead relies almost entirely on the use of guidance and orientation hypotheses. Hence, animals with hippocampal damage would not be at any particular disadvantage and should therefore perform on this task as well as normal animals. Finally, although orientation hypotheses are persistent, they can be modified by repeated nonreward, and evidence that supports this proposal is provided by the large numbers of responses that the hippocampal pigeons made to the unlit key on the correct side during the first three reversals only, their unlit correct key responses thereafter remaining at a fairly low and constant level.

It can be seen, therefore, that the main results of this experiment are again in good agreement with the cognitive mapping model of hippocampal function proposed by O'Keefe and Nadel, and they also support the proposal made earlier that the performance of the hippocampal pigeons in the probability task can be explained most readily in terms of impaired spatial ability, or spatial memory.

CHAPTER 5 Performance on a DRL 10 Schedule of Reinforcement

Introduction

As noted previously, hippocampal mammals frequently have been described as showing perseverative behaviour, in that there is a much greater tendency for these animals to repeat responses regardless of whether or not they are rewarded. Thus, hippocampal rats trained in different types of mazes repeat arm entries, either making particular sequences of responses repeatedly (Olton et al, 1978) or adopting a position habit (Isaacson et al, 1968). Related to this latter finding is the observation that hippocampal rats show reduced spontaneous alternation (Roberts et al, 1962). They also show impaired passive avoidance learning (Kimura, 1958), and greater resistance to extinction, especially in mazes and runways, although there have also been several reports of impaired extinction in an operant chamber (see Chapter 3, p.100).

On the basis of findings such as these, Clark and Isaacson (1965) suggested that animals with hippocampal lesions ought also to be impaired in their performance on operant schedules of reinforcement that require responses to be withheld from time to time in order to develop a temporal discrimination (see Chapter 1, p.47). One such task is the differential reinforcement of low rates of responding, or DRL, schedule. This requires animals to reduce their rates of responding in order to obtain reinforcement by effectively punishing responses that are made within a certain period following a reinforcement. This is achieved by the delay interval timer being reset each time a response is made during this interval. Thus, a DRL 20 secs schedule (usually abbreviated simply to DRL 20, and the most commonly used schedule in all of these studies) requires an animal to make a single response, which is rewarded, and then to refrain from responding for the next 20 secs, after which a reinforcement would again be available following the next response. An inability to withhold responses would therefore result, in the extreme case, in an animal receiving only the first reward of the session but

no subsequent rewards.

Clark and Isaacson did indeed find that hippocampal rats performed less efficiently on a DRL 20 schedule, showing higher rates of responding than the normal rats and consequently receiving fewer reinforcements. This they assumed was due to an inability to withhold responses during the delay interval, which in turn was the result of a greater resistance to extinction by the hippocampal rats. Subsequently others reported impaired DRL performance in hippocampal rats (Schmaltz and Isaacson, 1966a; Haddad and Rabe, 1967; Curtis and Nonneman, 1977) and monkeys (Jackson and Gergen, 1970). In all of these experiments the DRL training was preceded by a period of pretraining on a CRF schedule, and although Schmaltz and Isaacson (1966b) suggested that this pretraining was important for the occurrence of a DRL deficit in hippocampal animals, since lesioned rats not given CRF pretraining were not impaired on DRL performance, other reports by Ellen and his colleagues (Ellen, Wilson, and Powell, 1964; Ellen and Aitken, 1970; Ellen, Aitken and Walker, 1973), in which the rats were also given CRF pretraining, have suggested that both extensive CRF pretraining and large lesions are necessary for impaired performance on a DRL schedule by hippocampal animals. However, the results of an experiment by Johnson, Olton, Gage, and Jenko (1977) suggest that the discrepancies between the findings of Ellen et al and those of other investigators may be due more to the site of the lesion, having found that DRL deficits occurred in rats with anterodorsal hippocampal lesions, but not in those with posteroventral lesions.

Although the DRL schedule is initially a difficult task for rats to master, they are able eventually to perform efficiently, even on a DRL 60 schedule (see Kramer and Rilling, 1970 for a comprehensive review of DRL studies in normal animals). However, by comparison, pigeons are decidedly inferior and have great difficulty in adjusting to DRL schedules of 20 secs or longer (Powell, 1973). One important difference,

however, between the task for rats and the task for pigeons lies in the nature of the response required by the two species. Whereas the rat has difficulty in adapting to the schedule because some responses are rewarded while many are not, the pigeon, in addition, has to make the same response to the key as it does to obtain reinforcement. It is significant, in fact, that single-frame analysis of high-speed cine film of a pigeon's key-pecking responses has shown that, when food-reinforced, the pigeon pecked the key with a closed beak, but when water-reinforced, the key was pecked with an open beak (Jenkins and Moore, 1973). In order to pick up grains of food the pigeon has to close its beak so that grains are grasped by the tip, whereas, in drinking, pigeons siphon water, keeping their beaks open to do so. Thus, in pigeons, response topography also interferes with their acquisition of the task, and indeed Hemmes (1970 - cited in Schwartz and Williams, 1971) has shown that pigeons perform much more efficiently on a DRL 14 schedule when using a treadle-pressing response than when using the conventional key-pecking response.

While the pigeons in the first two experiments did not show a greater resistance to extinction compared with the normal pigeons, in both experiments they nevertheless did show a greater tendency to persist with particular response patterns and, more pertinently, in the second experiment they made significantly more responses to the unlit correct key during the first three reversals. This suggested, therefore, that pigeons with hippocampal lesions should be impaired in their ability to adapt to a DRL schedule. Since CRF pretraining appeared to be a necessary prerequisite for the occurrence of a DRL deficit, the pigeons from the previous experiment were used, and because of the difficulty that pigeons have with DRL schedules, a DRL 10 schedule was used here.

Method

Subjects

Twelve pigeons were used, six of which had received bilateral hippocampal lesions, the remaining six being sham-operated or unoperated controls, and they had all been trained on the serial position reversal task several months previously. They were maintained at 80% of their free-feeding weights, and water was freely available in their home cages.

Apparatus

A three-key operant chamber was used, in which the two side-keys were blanked off with pieces of grey card, matched as nearly as possible to the matt grey of the aluminium panel, and the centre key could be illuminated with white light.

Procedure

Pretraining

The pigeons had all received considerable CRF training in a discrete-trials task, but because of the time that had elapsed since the completion of the previous experiment, they were given three days of pretraining on a conventional CRF schedule, following key-peck retraining. Throughout each daily session the houselight and keylight remained on continuously, and although the response requirement was a single keypeck for each reinforcement, which consisted of 3 secs access to a grain mixture, all responses could be recorded. Each pigeon was run until it had obtained 100 reinforcements each day, and the time taken to achieve this was recorded, together with the total responses that were made.

DRL training

On the fourth day of the experiment the pigeons were transferred to the DRL 10 schedule. At the start of a session the first response was reinforced, but subsequently a response was only reinforced if 10 secs or more without a response had elapsed since

the end of the last reinforcement; responses during this 10 sec period merely served to reset the DRL timer and were not reinforced.

Each daily session was of 20 mins duration, the keylight and the houselight remaining on throughout, and reinforcement again was 3 secs access to grain. The end of a session was signalled by the keylight being switched off, and it also became inoperable and further responses could not be recorded. The pigeons were each given 20 days of DRL training, and on the day following the final session they were given a 20 mins extinction session, which was identical in all respects to the training sessions except that no responses were reinforced.

Each day, during DRL training, the total numbers of responses made and reinforcements obtained by each pigeon were recorded on electromechanical counters; in the extinction session the number of interresponse times (IRTs) greater that 10 secs were recorded in place of reinforcements. It had also been intended to record all IRTs, and in a manner which would readily allow computer analysis of the data; for these purposes an ADDO 4 paper tape punch was used. Unfortunately, however, as the experiment progressed the tape punch became increasingly unreliable, so that many of the shorter IRTs were recorded only sporadically. For this reason, therefore, any further attempts to record IRTs were abandoned and consequently an analysis of IRTs is not included here.

Results

Histology

Since the pigeons in this experiment had also been trained on the serial position reversal task, the histological data for these pigeons are presented in Chapter 4 (p.133).

Pretraining

Each of the pigeons required only minimal keypeck retraining, and they all responded readily on the CRF schedule, during which they all showed a strong tendency to overrespond. The numbers of responses made by the individual animals are presented

in Table 9, and are summarised in Figure 36. From these it can be seen that there were no differences between the two groups on this response measure. This was confirmed by an analysis of variance, which showed that there was no effect of the lesion treatment overall ($F(1,10) = 0.12$, $p = 0.74$). However, there was a significant effect over days ($F(2,20) = 10.99$, $p < 0.001$), showing that the numbers of responses decreased considerably from day 1 to day 3, but the interaction of lesion treatment x days was not significant ($F(2,20) = 0.05$, $p = 0.94$).

The times taken to obtain 100 reinforcements are presented in detail in Table 10 and are summarised in Figure 37, and it can be seen that the hippocampal group, although making approximately the same numbers of responses as the normal group on each of the three days, showed a tendency to respond faster in each CRF session. Analysis of variance confirmed this impression, showing that the hippocampal pigeons were significantly quicker in obtaining 100 reinforcements than the normal pigeons ($F(1,10) = 9.12$, $p = 0.013$), although neither the effect over days ($F(2,20) = 0.83$, $p = 0.45$), nor the groups x days interaction ($F(2,20) = 0.27$, $p = 0.72$) was significant.

The third measure used here was that of response-rate, and it was derived from the other two measures by dividing the number of responses in a session by the response time, which was obtained by deducting the total reinforcement time (300 secs) from the total time taken to complete 100 reinforced responses. These data for the individual subjects are presented in Table 11 and are summarised in Figure 38. It appears from this histogram that the response rate of the hippocampal group was noticeably higher on all three days, compared with the normal group, the response rate of the hippocampal animals on day 3 being marginally higher than that of the normal animals on day 1. However, an analysis of variance did not confirm this, but instead showed that, over the three days, the difference between the groups was not significant ($F(1,10) = 4.06$, $p = 0.069$). There was also no significant effect over days ($F(2,20) = 2.40$, $p = 0.12$), and the

Table 9

Total responses in CRF pretraining

Subjects	Normals Days 1	2	3	Hippocampals Days 1	2	3
1	197	104	100	182	118	104
2	186	139	106	365	172	104
3	266	154	122	217	101	103
4	361	100	102	297	217	133
5	103	106	100	102	100	100
6	101	109	103	100	101	101
Means	202.3	118.7	105.5	210.5	134.8	107.5

Table 10

Time (secs) for 100 reinforcements in CRF pretraining

Subjects	Normals Days 1	2	3	Hippocampals Days 1	2	3
1	430	439	506	392	380	362
2	492	392	565	383	377	368
3	580	380	395	388	348	379
4	416	387	375	412	415	402
5	528	509	527	415	370	349
6	396	550	388	500	450	396
Means	470.3	442.8	459.3	415.0	390.0	376.0

Table 11

Response rates in CRF Pretraining $\left(\frac{\text{Total responses}}{\text{Time for 100 reinforcements - 300 secs}}\right)$

Subjects	Normals Days			Hippocampals Days		
	1	2	3	1	2	3
1	1.52	0.75	0.49	1.98	1.48	1.68
2	1.08	1.51	0.40	4.40	2.23	1.53
3	0.95	1.93	1.28	2.52	2.10	1.30
4	3.11	1.15	1.36	2.65	1.89	1.30
5	0.45	0.51	0.44	0.89	1.43	2.04
6	1.05	0.44	1.17	0.50	0.67	1.05
Means	1.36	1.05	0.86	2.16	1.63	1.48

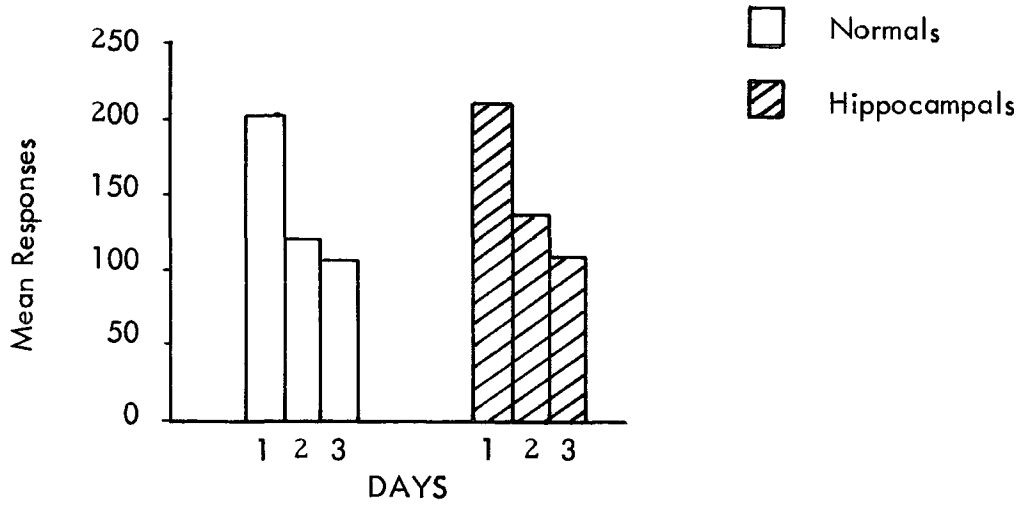


Figure 36. Mean responses made on each of the three days of CRF pretraining.

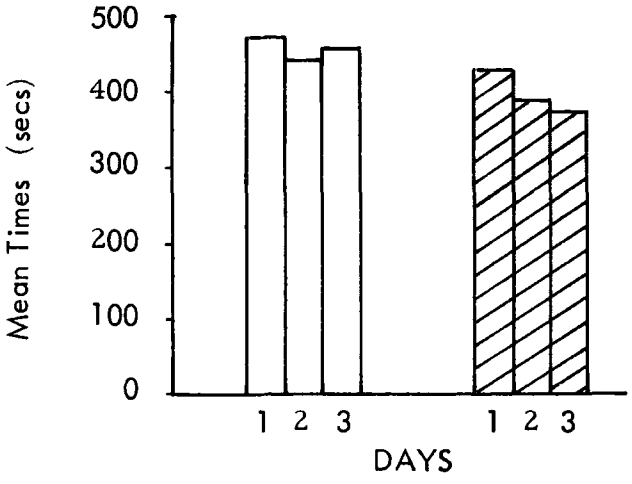


Figure 37. Mean times taken to obtain 100 reinforcements on each of the three days of CRF pretraining.

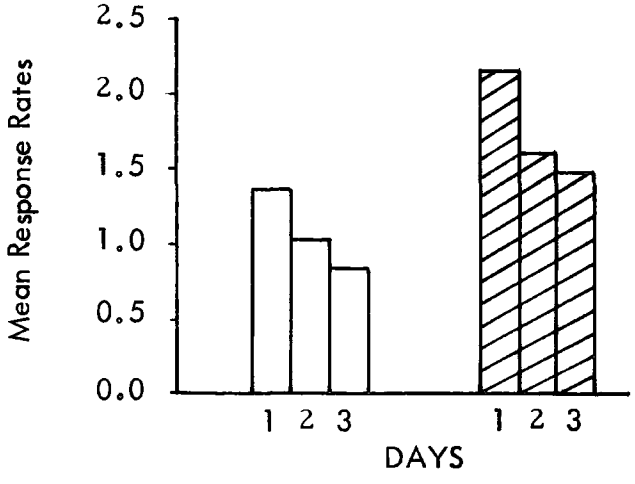


Figure 38. Mean response rates on each of the three days of CRF pretraining.

interaction between the groups over days was not significant ($F(2,20) = 0.08$, $p = 0.91$).

DRL training

All pigeons showed an immediate increase in their response rates when they were transferred from CRF to the DRL schedule, although this was more marked for the hippocampal group. The mean daily responses for each pigeon are presented in Figure 39, which shows that, following this initially higher rate of responding on day 1, both groups decreased their response rates considerably over the next two days. Apart from some fluctuation in their responses over days 4 - 6, the normal group further reduced the number of responses they made until they reached a level of responding on day 11 which they maintained for the next ten days. In contrast, the hippocampal group showed only a minimal reduction in responses after day 3, then maintaining a fairly constant level of responding, which was consistently higher than that of the normal group, for the remaining fourteen days of DRL training. An analysis of variance confirmed that the hippocampal pigeons made significantly more responses than the normal pigeons ($F(1,10) = 9.46$, $p < 0.012$), and that there was a significant reduction in responses over days ($F(19,190) = 12.66$, $p < 0.00005$), but it showed that the groups x days interaction was not significant ($F(19,190) = 1.05$, $p = 0.40$).

Figure 40 presents a summary of the mean numbers of reinforcements obtained daily by the two groups, and it can be seen that the hippocampal pigeons in fact gained more reinforcements on the first day than the normal pigeons did (means: normal group, 15.8; hippocampal group, 19.2) although this difference is not significant (Mann-Whitney $U = 13.5$, $p > 0.24$). However, whereas the numbers of reinforcements obtained by the normal pigeons then generally increased, the reinforcements obtained by the hippocampal pigeons showed a slight decline. When subjected to an analysis of variance, it was found that the number of reinforcements gained by the hippocampal group was significantly lower than the number gained by

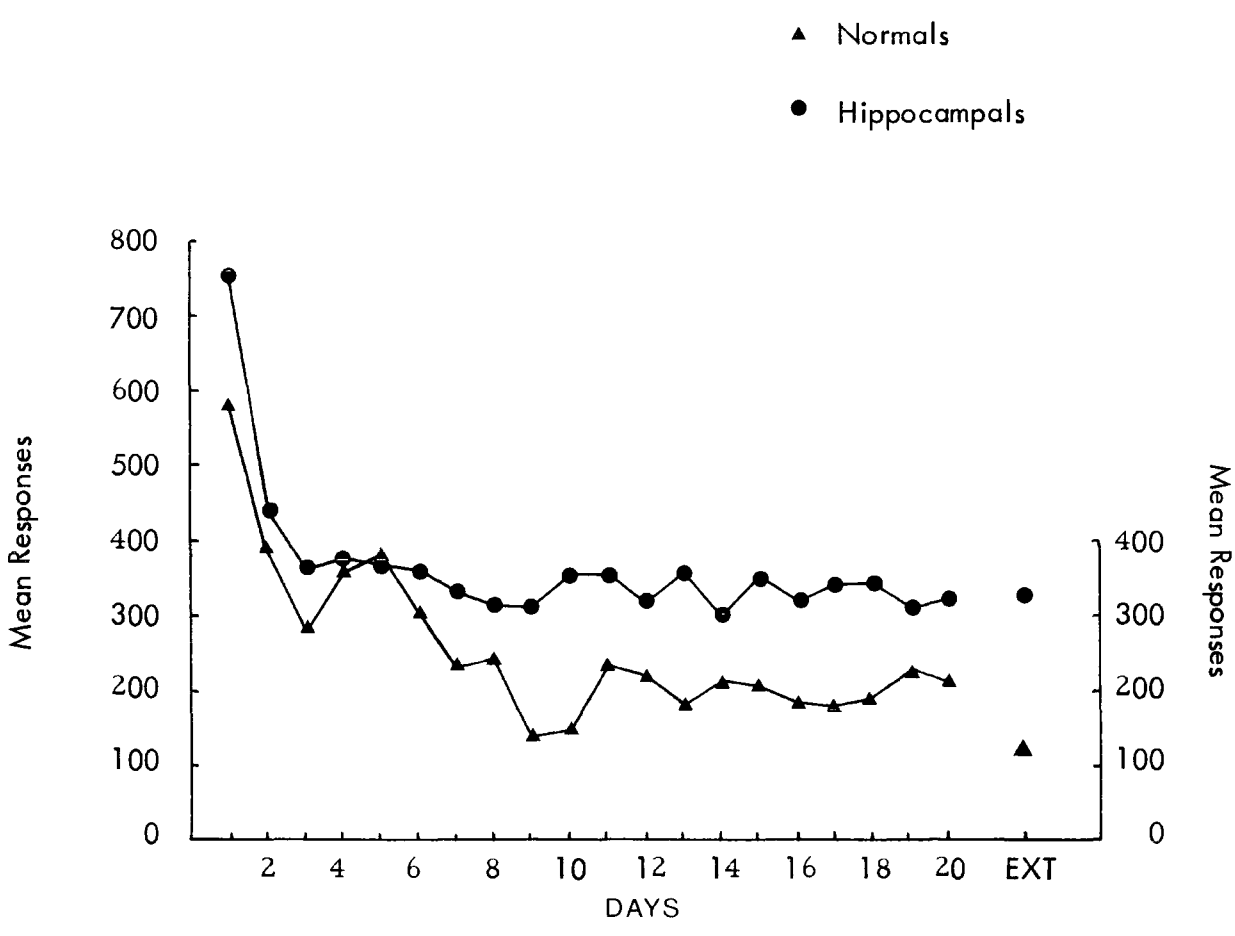


Figure 39. The mean numbers of responses made over the 20 days of DRL 10 training, and in the single extinction session (EXT).

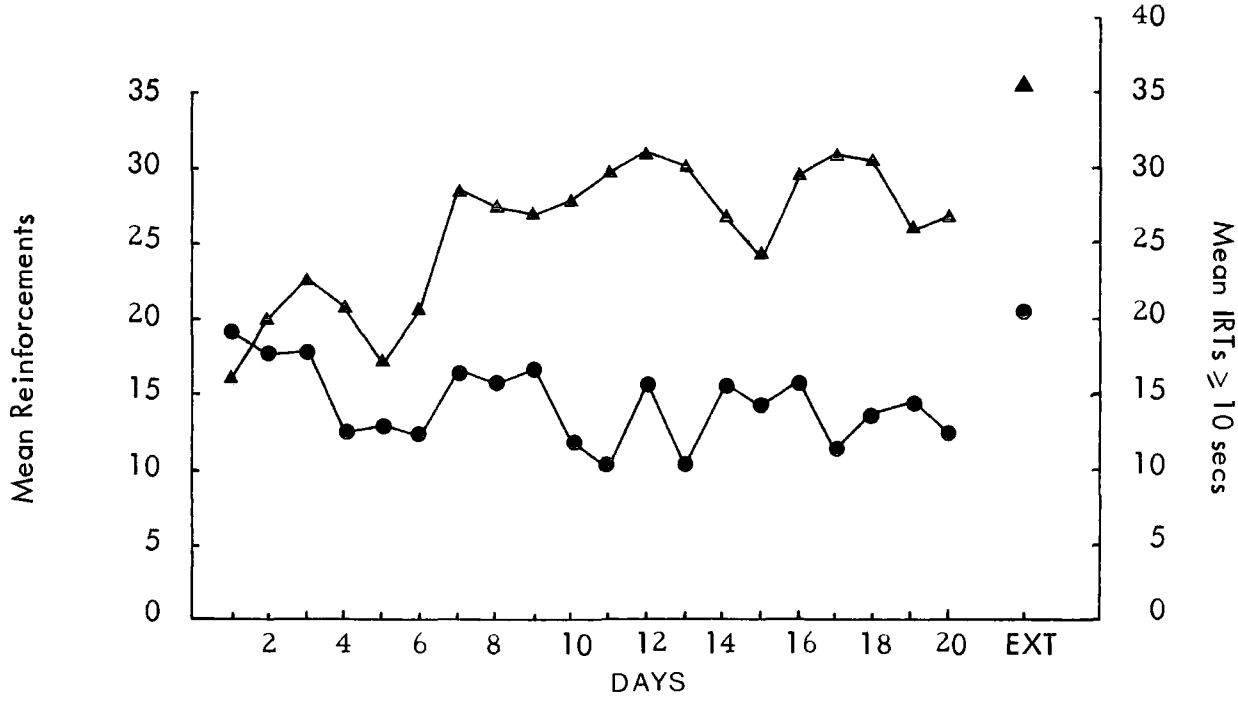


Figure 40. The mean numbers of reinforcements obtained over the 20 days of DRL 10 training, and the number of IRTs $\geq$ 10 secs in the single extinction session (EXT).

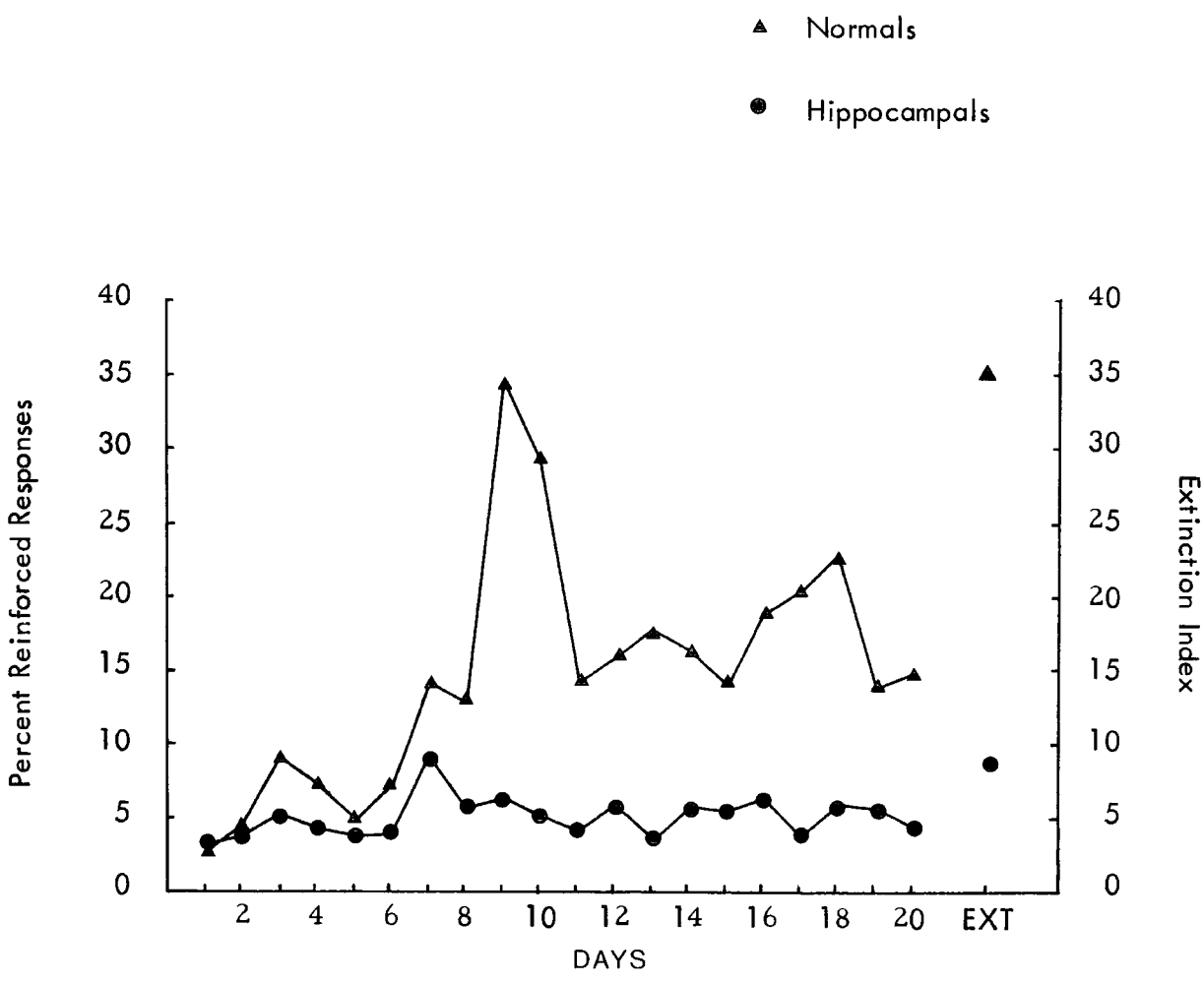


Figure 41. The mean percentage of reinforced responses over the 20 days of DRL 10 training, and the mean extinction index in the single extinction session (EXT).

the normal group ($F(1,10) = 14.43$, $p < 0.004$). Although there was not a significant effect over days ($F(19,190) = 1.44$, $p = 0.11$), there was found to be a significant interaction between the two groups over days ($F(19,190) = 2.61$, $p < 0.001$), showing that the normal group did increase the number of reinforcements they obtained more than the hippocampal group did.

In common with many other studies of DRL responding (see Kramer and Rilling, 1970), the third measure that was used here was the percentage of reinforced responses, or efficiency ratio, obtained by dividing the number of reinforcements gained by the total number of responses made in a session, and this is summarised in Figure 41. This shows that the percentage of reinforced responses made by the hippocampal pigeons was extremely low and remained virtually constant over the twenty days of DRL training. On the other hand, the normal pigeons, who also began by responding at the same low level of efficiency, showed a marked improvement in performance after the fifth day, and although this was not maintained, they nevertheless made approximately three times the proportion of reinforced responses that the hippocampal pigeons made (mean efficiency ratios: normal group, 14.8%; hippocampal group, 5.1%). The analysis of variance that was carried out on these data confirmed that the efficiency scores of the hippocampal pigeons were significantly lower than those of the normal pigeons ($F(1,10) = 14.59$, $p < 0.004$); that together, the two groups showed a significant increase in efficiency over the twenty days ($F(19,190) = 3.01$, $p = 0.0001$); and that the groups x days interaction was also significant ($F(19,190), = 2.39$, $p < 0.002$), thereby showing that the performance of the normal group improved much more than did that of the hippocampal group.

Extinction

The three measures that were used in DRL training were also used to indicate extinction performance, except that, because responses were not reinforced during

extinction, IRTs of $\geqslant 10$ secs were recorded instead, and consequently the measure that is equivalent to the efficiency ratio during training, and which here is referred to as the extinction index,was obtained by expressing the number of IRTs $\geqslant 10$ secs as a percentage of the total responses. These extinction measures, which can then be directly compared with the scores obtained during DRL training, are presented for the individual animals in Table 12, and the means in each case for the two groups are plotted on the appropriate graphs immediately following the final day of training. By referring to Figure 39 it can be seen that in extinction the normal group made slightly more than half the number of responses that they made on the last day of DRL training, thereby showing a reliable reduction in response rate. As a consequence of this reduced response rate they achieved a greater number of IRTs $\geqslant 10$ secs than they did on any of the training sessions (see Figure 40), and thus they obtained a fairly high extinction index, indicated in Figure 41.

In contrast, the hippocampal pigeons maintained the number of responses they made at the end of DRL training, showing, therefore, no reduction in response rate during extinction. However, it can also be seen, from Figure 40, that they achieved a greater number of IRTs $\geqslant 10$ secs than they had done during training, and consequently their extinction index was higher than their efficiency ratio was on the final day of DRL training. Thus, although the hippocampal pigeons did not show any reduction in the total responses that they made, they nevertheless were able to reduce their overall response rate, so that the distribution of their IRTs shifted from primarily shorter to longer values. Nevertheless, on all three measures the hippocampal pigeons were noticeably impaired in this extinction task compared with the normal pigeons, and the differences between the two groups were found to be significantly different using the Mann-Whitney U test (responses: $U=3$, $p=0.016$; IRTs $\geqslant 10$ secs: $U=4.5$, $0.042>p>0.026$; extinction index: $U=2$, $p=0.004$).

Table 12

Extinction session data

Subjects	Normal			Hippocampal		
	Responses	⩾ 10 sec IRTs	Extinction Index	Responses	⩾ 10 sec IRTs	Extinction Index
1	51	24	47.1	243	22	9.1
2	90	44	48.9	166	40	24.1
3	95	27	28.4	288	12	4.2
4	73	35	48.0	457	8	1.8
5	299	46	15.4	322	24	7.5
6	156	36	23.1	449	19	4.2
Means	127.3	35.8	35.1	320.8	20.8	8.5

Discussion

Clark and Isaacson (1965) reported an impression that their hippocampal rats acquired the lever-press response more readily than either the neocortical or the unoperated control animals, although the hippocampal rats responded at a lower rate than the unoperated controls. Subsequently, Schmaltz and Isaacson (1966b) tested this idea by obtaining two measures of performance during the shaping trials, in which responses were reinforced on a CRF schedule, and found that the hippocampal animals spent less time and required fewer reinforcements than the unoperated controls to reach some shaping criterion. Thus, hippocampal rats acquire CRF responding faster than normal rats. Similarly, Means, Walker, and Isaacson (1970) found that hippocampal rats trained on a CRF schedule obtained 200 reinforcements faster than the control rats did. These results are therefore very similar to those obtained in the present experiment, in which it was found that in the pretraining period, the hippocampal pigeons obtained 100 reinforcements more quickly than the normal pigeons, but whereas Clark and Isaacson (1965) reported a lower response rate in the hippocampal rats, the hippocampal pigeons in this experiment did not differ from the normal pigeons in their response rates.

The major finding in this experiment, however, was that the pigeons with hippocampal lesions were decidedly inferior to the normal pigeons in their ability to adapt to a DRL 10 schedule, making many more responses and consequently receiving considerably fewer reinforcements. These results are therefore consistent with the majority of those reported in studies of the effects of hippocampal lesions in rats (Clark and Isaacson, 1965; Schmaltz, Wolf, and Trejo, 1973; Curtis, and Nonneman, (1977) and monkeys (Jackson and Gergen, 1970) on a DRL schedule. As noted in the Introduction to this experiment, the majority of these studies have used a DRL 20 schedule, although different daily training periods have been used. Nevertheless, the efficiency ratio is a measure which allows comparison between different experiments,

and in many of these the normal animals were capable of achieving efficiency ratios of between 35% and 55%, the squirrel monkeys in the experiment by Jackson and Gergen reaching a level of 50% reinforced responses, and therefore not being superior to some of the rats (e.g., Curtis and Nonneman, 1977, whose rats achieved 55% efficiency). In contrast, the hippocampal animals obtained between 5% and 15% reinforced responses, the majority being in the lower half of this range. In comparison with these, the present results showed that the normal pigeons were capable of obtaining nearly 35% reinforced responses, although their average over the 20 days was less than half this. On the other hand the hippocampal pigeons achieved approximately 5% reinforced responses, a value that is therefore similar to that obtained by many hippocampal rats. Furthermore, a characteristic of many of the mammalian studies is that, whereas the normal animals improved their performance over days, the hippocampal rats and monkeys showed very little change. Again, the results of the present experiment are consistent with this finding. However, the present results, in common with much of the mammalian hippocampal data, are not in agreement with the earlier reports of Ellen et al (1964, 1970). More recently Ellen et al (1973) have argued that both extensive CRF training and large lesions are necessary for DRL deficits to occur. In the present experiment, although three days of training on a CRF schedule in order to obtain 100 reinforcements each day would appear to constitute rather limited CRF training, certainly in comparison with the 10 - 20 days of CRF training for 150 reinforcements per day that the rats of Ellen et al (1970, 1973) received (and roughly equivalent amounts of CRF training also appear to have been given to the rats in the experiments by Clark and Isaacson, 1965; Schmaltz and Isaacson, 1966a, 1966b), given their previous experimental history, it would not seem unreasonable to assume that the pigeons had also received extensive CRF training. However, the question of lesion size is more difficult to deal with. It would appear from the histological reconstructions of the

lesions (see Chapter 4, p.133) that they were, in fact, moderate, but as the avian hippocampus is not the prominent and clearly-defined structure that it is in the mammalian brain, it would seem difficult at the present to attempt to estimate the size of the hippocampal lesion (as opposed to the extent of the total brain lesion).

The results of the single extinction session are of interest, not only because they showed there to be an extinction deficit in the hippocampal pigeons, whereas in the first two experiments extinction deficits did not occur, but also because the pigeons in the present experiment had been trained on the previous experiment, thereby showing that they were capable of normal extinction performance under certain conditions, but not under others. This clearly provides good evidence in support of the proposal made earlier that an increased resistance to extinction cannot be explained in terms of an impaired ability to withhold responses, and further, shows the nature of the task to be an important factor. Evidence that provides further support for this comes from an experiment by Kelsey and Grossman (1971). Ellen et al (1964) had shown that rats with septal lesions were impaired on a DRL schedule following CRF training. But Kelsey and Grossman, using a modified DRL task in a runway involving two response levers, one in each goal-box, with the requirement that responses are made on alternate levers on a DRL 30 schedule, found that, compared with normal rats, septal rats made significantly fewer perseverative errors on the lever on which they had just obtained a reward, but made significantly more errors on the other lever in anticipation of the potentially available reward.

The present results would appear to differ from those obtained by Nonneman et al (1974), who report that, following training on a DRL 20 schedule, on which they were impaired, hippocampal rats were then not impaired on extinction. However, Nonneman et al did find that their hippocampal rats made considerably more responses in each of the five half-hour extinction sessions compared with either the sham-operated control

rats or rats with lesions of the prefrontal cortex. But, they argued, because the hippocampal rats showed a significantly higher response rate during DRL training, the various groups could be compared only by calculating the response rates during extinction relative to the response rates on the final day of DRL 20 training. When this was done they found that the hippocampal rats were no longer impaired. But a similar calculation carried out on the present data shows that the hippocampal pigeons still made significantly more relative responses in extinction than the normal pigeons (Mann-Whitney $U=4$, $p=0.013$).

However, Kearsley, Van Hartesfeldt, and Woodruff (1974) also found that, following DRL 20 training, groups of male and female hippocampal rats made significantly more responses than normal rats during the first three days of extinction trials, and they took this to indicate a deficit in performance. Unfortunately, however, the only other indicator of extinction performance that they present is a measure of the decline in the response rate over days. It is not possible, therefore, to make any further comparisons between their results and those from the present experiment. However, it is clear that the present results are, in fact, in agreement with those of Nonneman et al and of Kearsley et al, as far as the finding of a higher response rate by the hippocampal animals on the first day of extinction is concerned (and, as far as the writer is aware, these are the only two studies in which extinction trials were given to hippocampal animals following DRL training).

The fact that the hippocampal pigeons were impaired in extinction following the DRL schedule, but not after spatial reversal training, was attributed earlier to task differences. Since the hippocampal pigeons made a large number of responses during DRL training, but received relatively few reinforcements, the most likely explanation for their higher response rate in the single extinction session, compared with the normal pigeons, would seem to be that they treated this extinction session as the same

as the previous DRL schedule. For the hippocampal pigeons, the reduction in the number of reinforcements they obtained between the last DRL session and the extinction session was somewhat less than that experienced by the normal pigeons.

This would appear to be another example of the persistence of particular responses patterns or strategies that has been reported in hippocampal mammals (see the Introduction to this experiment) and which has also been found to occur in hippocampal pigeons in other tasks (see Chapters 3 and 4). The various results of the present experiment are consistent with those that have been reported in similar experiments on hippocampal rats and monkeys, and they therefore provide further support to the proposal that the avian and the mammalian hippocampus are functionally, as well as structurally, similar.

CHAPTER 6 The Effects of Overtraining on the Reversal of a Visual Form Discrimination

Introduction

Although there have been many reports of impaired reversal learning in mammals with hippocampal lesions, by far the greater proportion of deficits have been found to occur in spatial discriminations (see Chapter 4). Furthermore, while there have been a number of studies in which hippocampal lesions were found to impair the reversal of a nonspatial discrimination in rats (Silveira and Kimble, 1968), cats (Teitelbaum, 1964; Webster and Voneida, 1964; Nonneman and Isaacson, 1973) and monkeys (Douglas and Pribram, 1966), there have been as many reports in which hippocampal mammals were not impaired (rats: Samuels, 1972; cats: Isaacson et al, 1968; monkeys: Mahut, 1971; Jones and Mishkin, 1972), or were superior to normals (monkeys: Schram, 1970; Zola and Mahut, 1973). Moreover, Mahut (1971), Jones and Mishkin (1972), and Samuels (1972) found that, although hippocampal animals were unimpaired on the reversal of a nonspatial discrimination, the same animals were impaired on the reversal of a spatial discrimination. These findings strongly suggested the importance of spatial factors in the hippocampal deficit, and this observation has already been made by Mahut (1971) and Samuels (1972), as was noted in the Introduction in Chapter 4. In the experiments reported in Chapters 3 and 4 of this thesis it was found that pigeons with hippocampal lesions performed more efficiently than normal pigeons on the colour probability task, but were impaired on the serial position reversal task, and the data showed that these effects were mainly due to inconsistent responding to spatial cues by the hippocampal animals. It was argued, therefore, that the behavioural effects of hippocampal lesions in pigeons were similar to those in mammals. Clearly, additional support for this proposal would be gained from the finding that, although hippocampal pigeons showed a deficit on a spatial reversal task, they were not necessarily impaired on a nonspatial reversal task.

However, a further finding that was felt to be of interest was the differential effects

of overtraining on reversal learning in rats and birds (Sutherland and Mackintosh, 1971). If, once rats reach criterion on the acquisition of a visual discrimination, they are given sufficient extra (overtraining) trials, it is found that they learn the reversal of the original task faster than rats not given overtraining trials. This has been called the overtraining reversal effect (ORE), and it appears that, although overtraining increases resistance to extinction of responses to the previously correct stimulus (S+), its main effect on reversal performance is to reduce the number of trials the rat spends in responding to position (Mackintosh, 1969). However, it more often than not has been found that the ORE does not occur in rats trained on a spatial discrimination, and Sutherland and Mackintosh (1971) have suggested that this may be due to the rat's natural preference for spatial cues. Furthermore, they had suggested that, if this were the case, then birds, whose natural preference would appear to be for visual cues, should not show an ORE when trained on a visual discrimination. Evidence supporting this came from an experiment by Brookshire, Warren, and Ball (1961) in which they found that overtraining on a brightness task facilitated reversal learning in rats but retarded it in chicks. This experiment was repeated by Mackintosh (1965), who obtained a similar result. However, when chicks were trained on a more difficult visual task (a shape discrimination), overtraining no longer impaired reversal learning, but neither did it facilitate it (Schade and Bitterman, 1965), and Mackintosh (1965) found that when chicks were trained on an extremely difficult orientation discrimination, with both position and brightness irrelevant, a significant ORE did occur. Mackintosh also found that, whereas overtraining has a facilitatory effect in rats because it reduces position responding, it had the opposite effect in chicks trained on an easy discrimination, such as brightness, because it increased position responding, in addition to increasing resistance to extinction. A similar effect was also reported by Matyniak and Stettner (1970) in pigeons trained on a simultaneous visual discrimination involving three white

horizontal or vertical stripes on a black background. The finding that, unlike normal pigeons, hippocampal pigeons were unable to respond consistently to spatial cues suggested, therefore, that overtraining on a relatively easy visual discrimination should not increase position responding in hippocampal pigeons to the same extent that it does in normal pigeons.

Finally, by measuring response latencies during the acquisition of a simultaneous visual discrimination, Olton (1972a) found that hippocampal rats clearly were discriminating between the two stimuli in terms of different response latencies to S+ and S-, even though they were still responding consistently to their preferred position. However, the hippocampal rats maintained their position habit for much longer than the normal rats, and Olton therefore proposed that hippocampal rats were not impaired in their ability to suppress responses, but that they were impaired in their ability to shift responses. Although the evidence obtained from an analysis of the sequences of responses in the serial position reversals task (Chapter 4) showed that the hippocampal pigeons were not suffering from a response-shift deficit, it nevertheless was felt that an analysis of response latencies on both the acquisition and the reversal of a simultaneous visual discrimination might provide further information concerning the response strategies of hippocampal pigeons during discrimination learning, since there is evidence (Kimble and Kimble, 1970) that, although hippocampal rats are capable of learning a visual discrimination at a normal rate, their use of particular hypotheses is different from that of normal rats.

The present experiment was therefore designed primarily to investigate the effects of hippocampal lesions in pigeons on the acquisition and reversal of a nonspatial discrimination. In addition, because of their impaired ability to respond to spatial cues, it was expected that overtraining would not retard reversal learning in hippocampal pigeons trained on a relatively simple visual discrimination in the way that it would in

normal pigeons. On the other hand, it follows that there should be no difference between the reversal performance of normal and hippocampal pigeons when they are overtrained on a more difficult visual discrimination. However, a preliminary study in a small group of normal pigeons suggested that the horizontal-vertical discrimination that was used here was a relatively simple task.

Method

Subjects

Twenty-four pigeons were used. They were maintained at approximately 80% of their ad lib weight, and water was freely available in their home cages. Half of the animals were given bilateral hippocampal lesions and the other half were either sham-operated or unoperated controls. The pigeons were also tested in two other experiments, a delayed spatial alternation task and a delayed colour alternation task (see Chapters 7 and 8 respectively), which, together with the present experiment, were run in a balanced design.

Surgery

Full details of the surgical procedures involved are presented in Chapter 2.

Apparatus

In this experiment a three-key operant chamber was used. The two side keys were fitted with miniature inline stimulus projectors, by means of which either white light or simple pattern stimuli could be back-projected on to the keys, but the centre key could be illuminated only with white light. The stimuli used were a black horizontal bar and a black vertical bar, each 2 mm wide and 28 mm long, on a white background.

Procedure

Experimental design

All 24 pigeons were first pretrained to keypeck and to obtain food reward (see

below). They were then randomly assigned to three groups of eight pigeons, each consisting of four hippocampal and four normal subjects. The pigeons were assigned to the three experiments as shown in Table 13, and the three groups were run in the three experiments in the order

A B C

B C A

C A B

SUBJECTS N	SUBJECTS H	ORDER OF TESTING	EXPERIMENT
76	73	A1	Acquisition and
88	79	(B2)	reversal of a
90	86	(C3)	visual form
92	91		discrimination
78	75	B1	Delayed spatial
80	83	(C2)	alternation
96	87	(A3)	
108	95		
81	85	C1	Delayed colour
82	89	(A2)	alternation
84	98	(B3)	
109	137		

Table 13 Experimental design

Pretraining

Pretraining began 7-10 days postoperatively, and all pigeons were first habituated to the apparatus and trained to obtain food from the food hopper whenever it was presented, as described in Chapter 2. They were then autoshaped to peck at any of

the three keys when they were illuminated with white light (Brown and Jenkins, 1968). The centre key was lit on the first trial of each day, the two side keys remaining blank, and subsequently on every third trial; the intervening trials were presented on the two side keys according to a Gellerman sequence (Gellerman, 1933). Five different Gellerman sequences were used, one for each daily session, and these are included in the Appendix. A keylight was presented for 8 secs, or less if the key was pecked once during this period, when it was extinguished and the food hopper presented for 3 secs. This was then followed by a variable intertrial interval (ITI), with a mean of 40 secs and a range of 10-80 secs. The houselight remained on throughout the session. The sequence of ITIs used was 20, 60, 40, 10, 80, 30, 20, 50, 15, 35, 80, 40 secs, and each session consisted of 30 trials in which all three keys were presented, each for a total of 10 trials. The maximum duration of a session was approximately 25 mins, and consequently only half the pigeons were run each day; thus each pigeon was run on alternate days. All subjects were trained until they reached a criterion of 7 or more responses on each key in a daily block of 30 trials. The following day they were given a 30 trial session in which the requirement was now three responses per key (FR3), and on the next day a further 30 trials in which the response requirement was FR5.

After completion either of pretraining or of the delayed colour alternation task, half of the hippocampal animals and half of the normal animals in each group were randomly assigned to the non-overtrained condition in the present experiment, while the remaining animals were assigned to the overtrained condition. Thus, in this experiment there were four groups of six pigeons each:

I. Normals non-overtrained (NR)
II. Normals overtrained (NO)
III. Hippocampals non-overtrained (HR)
IV. Hippocampals overtrained (HO)

Training

Each training session began with the houselight on and the centre key illuminated with white light. A single response to the centre key switched off the keylight and presented the side keys, on one of which was projected the horizontal bar, and on the other the vertical bar. For half of the animals the horizontal bar was the positive stimulus, and for the other half the vertical bar was positive, and the spatial presentation of the two stimuli was determined by Gellerman sequences (see Appendix) which were punched on to paper tape and fed through a tape reader. A discrete trials procedure was used in which the response requirement was 5 responses (FR5) to either side key, which then caused both keylights to be extinguished. Correct and incorrect responses were counted separately so that whether the trial was correct or incorrect was determined by the key on which the FR5 requirement was first reached. Responses on the correct key resulted in 3 secs access to food, while 5 responses on the incorrect key instead turned off the houselight for 3 secs timeout (TO). Either event was then followed by a 5 secs ITI, during which the houselight was on but the keylights remained off. At the end of the ITI the two predetermined counters used to control the FR5 schedule on the two side keys were automatically reset and the white centre keylight came on again to signal the start of the next trial. All pigeons were given 50 trials per day and were trained to a criterion of 9 out of 10 correct on two consecutive blocks of 10 trials.

On the day following criterion performance on the acquisition of the discrimination, the pigeons assigned to the non-overtrained groups were reversed, so that the previously positive stimulus was now the negative stimulus, while the remaining pigeons were each given 500 overtraining trials before beginning reversal learning. As in acquisition, the criterion on the reversal task was 9 correct responses in 10 trials on two consecutive blocks of 10 trials.

For each daily session total correct, incorrect, left, and right responses were recorded on electromechanical counters for each subject. A Sodeco printout counter was also used to record trial by trial sequences of correct and incorrect responses so that, by comparing them with the appropriate stimulus sequences, the sequences of left and right responses could be determined for individual pigeons. A second printout counter, driven by a 0.1 sec multivibrator, was used to record the response latencies, defined as the time between the onset of the side-keys and the completion of the FR5 requirement on one of them. On completion of the experiment the response choice and response latency data were transferred manually on to data files on the departmental PDP8E computer, together with the five Gellerman sequences that were used in the acquisition and reversal training, so that the relatively large amount of trial by trial data that was obtained from this experiment could be analysed in detail.

Results

Histology

The reconstructions of the lesions that were produced in the twelve experimental pigeons used in this and the following three experiments are presented in Figure 42. (The final experiment, presented in Chapter 9, was a DRL 10 task in which six hippocampal and six normal pigeons, chosen randomly from the twenty-four pigeons used here, were tested several months after the completion of the three experiments that were involved in the balanced design described here.) The lesions extended, variously, from A8.50 to A3.25, but were mainly in the region A7.00 to A3.50. The smallest lesions occurred in pigeon No. 79, and tended to be slightly displaced laterally, but nevertheless involved small amounts of damage to the hippocampus pars dorsalis and to the hippocampus. Relatively small lesions also occurred in pigeons No. 89 and No. 91. The largest lesions were produced in pigeon No. 137, extending from A8.50 to A3.25 and involved damage mainly to the hippocampus pars dorsalis and to APH,

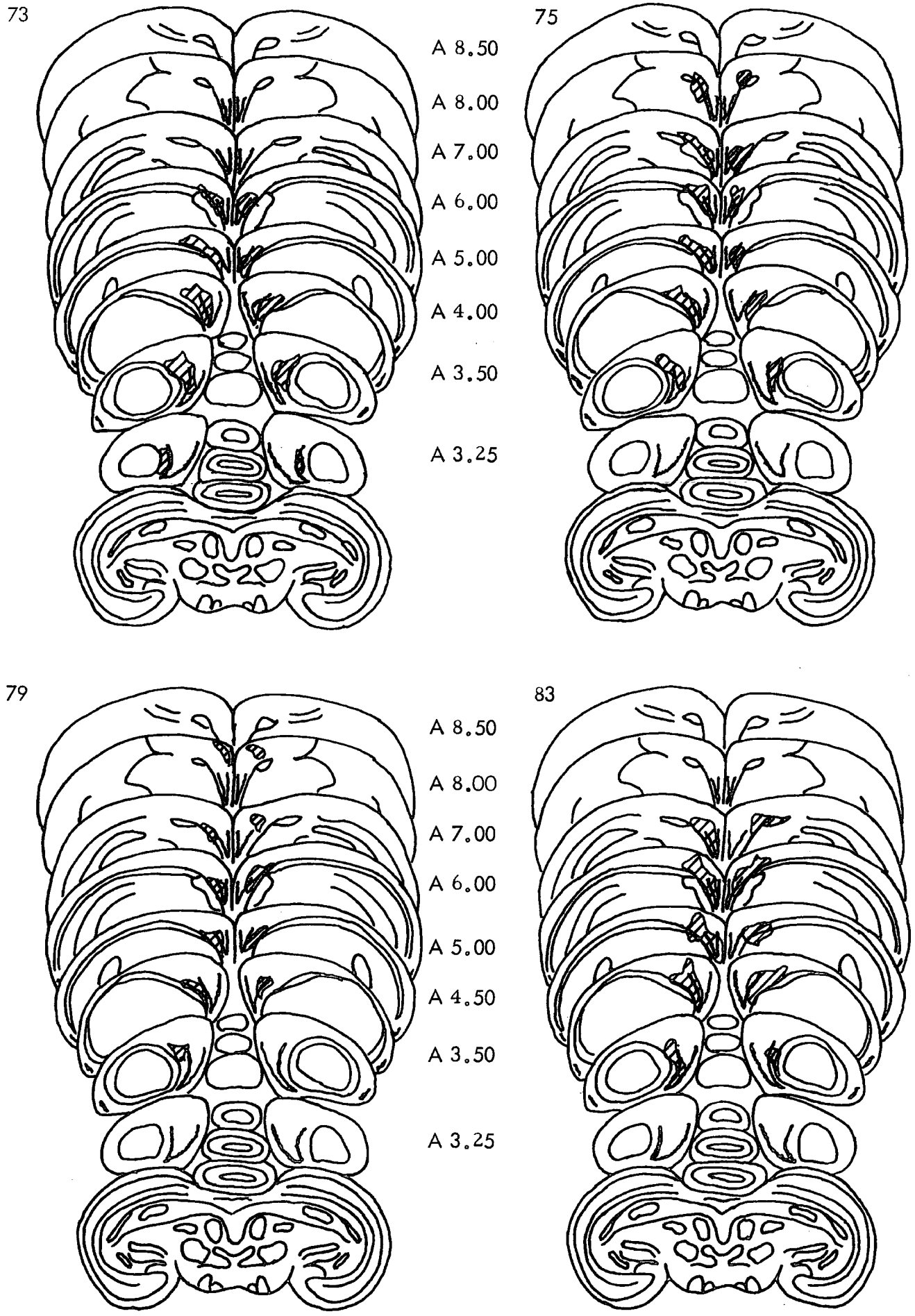


Figure 42. Reconstructions of the hippocampal lesions in the experimental pigeons that were used in the present experiment and in the experiments presented in Chapters 7, 8, and 9, based on the stereotaxic atlas of Karten and Hodos (1967).

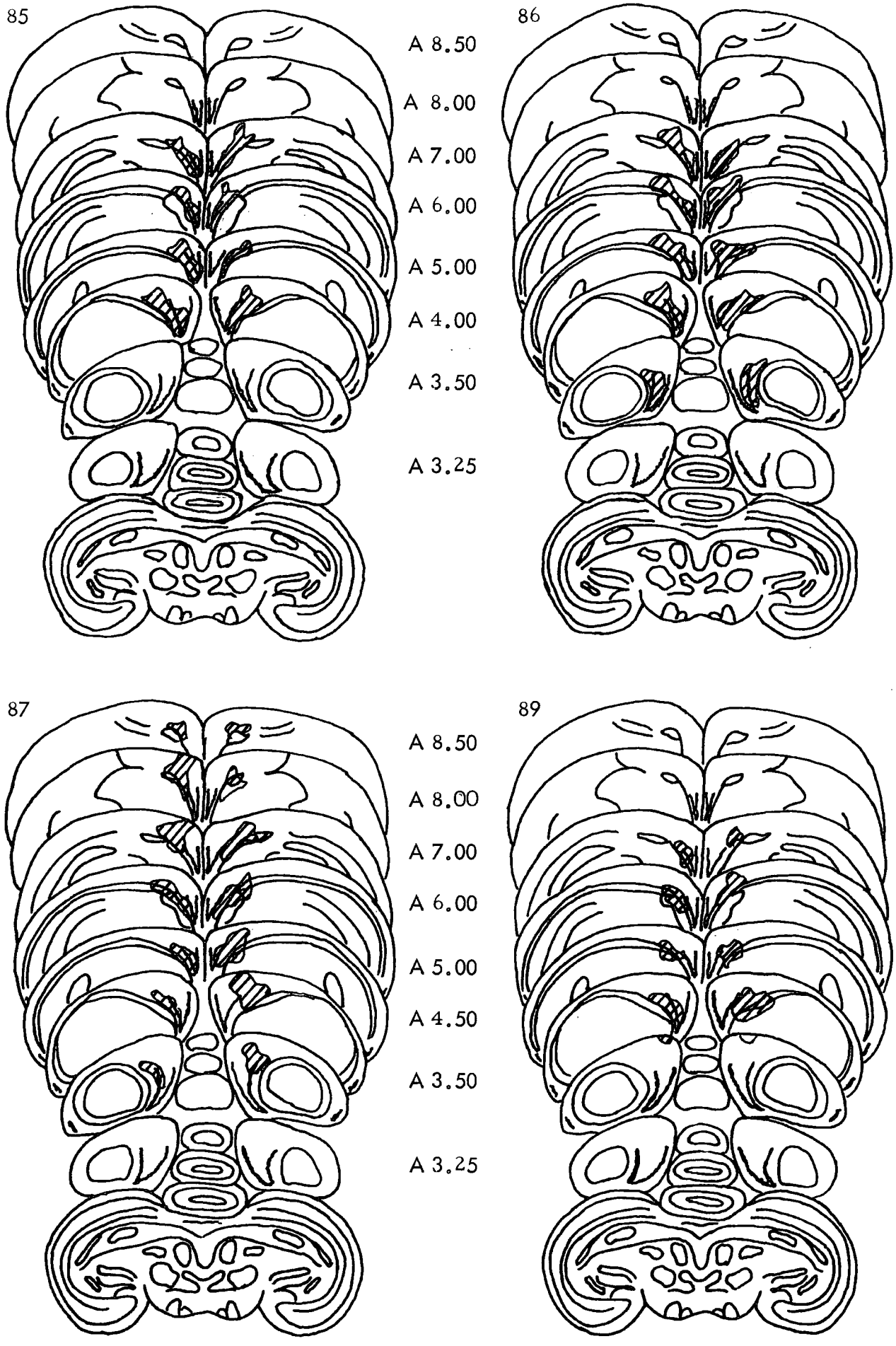


Figure 42 (contd.)

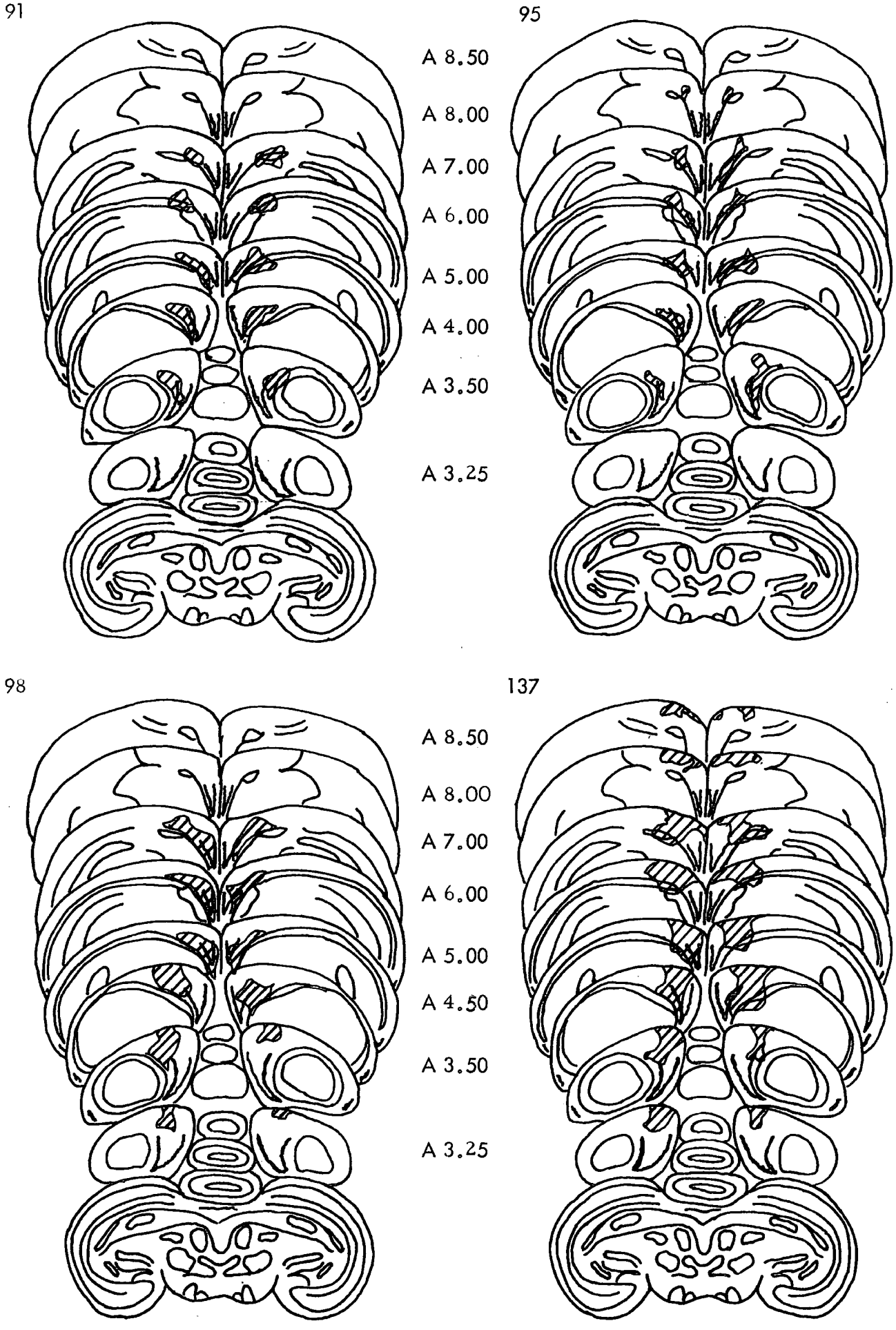


Figure 42 (contd.)

although some damage also occurred in the hippocampus in the region A 5.00 to A 3.50. In the majority of pigeons, however, the lesions were predominantly in the hippocampus and hippocampus pars dorsalis, and also small to moderate amounts of damage were invariably found in the parahippocampal area. In addition, small invasions occurred in the extreme posterior region of the accessory hyperstriatum (A 8.50 to A 8.00) in three pigeons (Nos. 75, 87, and 137), and minimal damage in the ventral hyperstriatum and caudal neostriatum was found in the majority of the pigeons. Again, the lesions produced in these pigeons appear to be quite consistent with those produced in the pigeons that were used in the previous experiments.

Pretraining

The data that were obtained from the autoshaping procedure are presented for individual pigeons in Tables 14 and 15, and are summarised in Figure 43. As the histogram shows, the hippocampal pigeons took marginally fewer trials, made marginally fewer errors to criterion (an error being defined here as a failure to make a response on a trial), and took slightly fewer trials to make their first response to the lighted key, compared with the normal pigeons. Unrelated t tests on each of these three measures confirmed that none of the differences between the two groups was significant (in each case, $t<1$, $df=22$, $p>0.4$).

Training

Since the twenty-four pigeons had been divided into three subgroups (A1, A2, and A3) immediately following pretraining, and two of the subgroups were trained in either one or two other experiments before being trained on the present task, it was important to determine whether the order in which the pigeons had been trained had had any systematic effect on their performance on the present task. A four-factor repeated measures analysis of variance (Keppel, 1973, p. 457), in which the factors were lesion treatment x overtraining x order of training x acquisition/reversal, was

Table 14

Trials and errors to criterion in autoshaping

	Trials		Errors	
	Normals	Hippocampals	Normals	Hippocampals
	180	90	141	56
	90	120	65	76
	150	150	124	75
	90	60	56	36
	90	90	39	51
	120	90	59	58
	60	60	23	25
	120	120	86	82
	90	150	25	107
	120	90	70	27
	120	120	52	68
	120	120	75	44
Means	112.5	105.0	67.9	58.8

Table 15

Trials to first response in autoshaping

	Normals	Hippocampals
	127	55
	21	9
	30	25
	21	35
	37	10
	31	31
	4	19
	73	73
	4	100
	1	10
	1	1
	72	1
Means	35.2	30.8

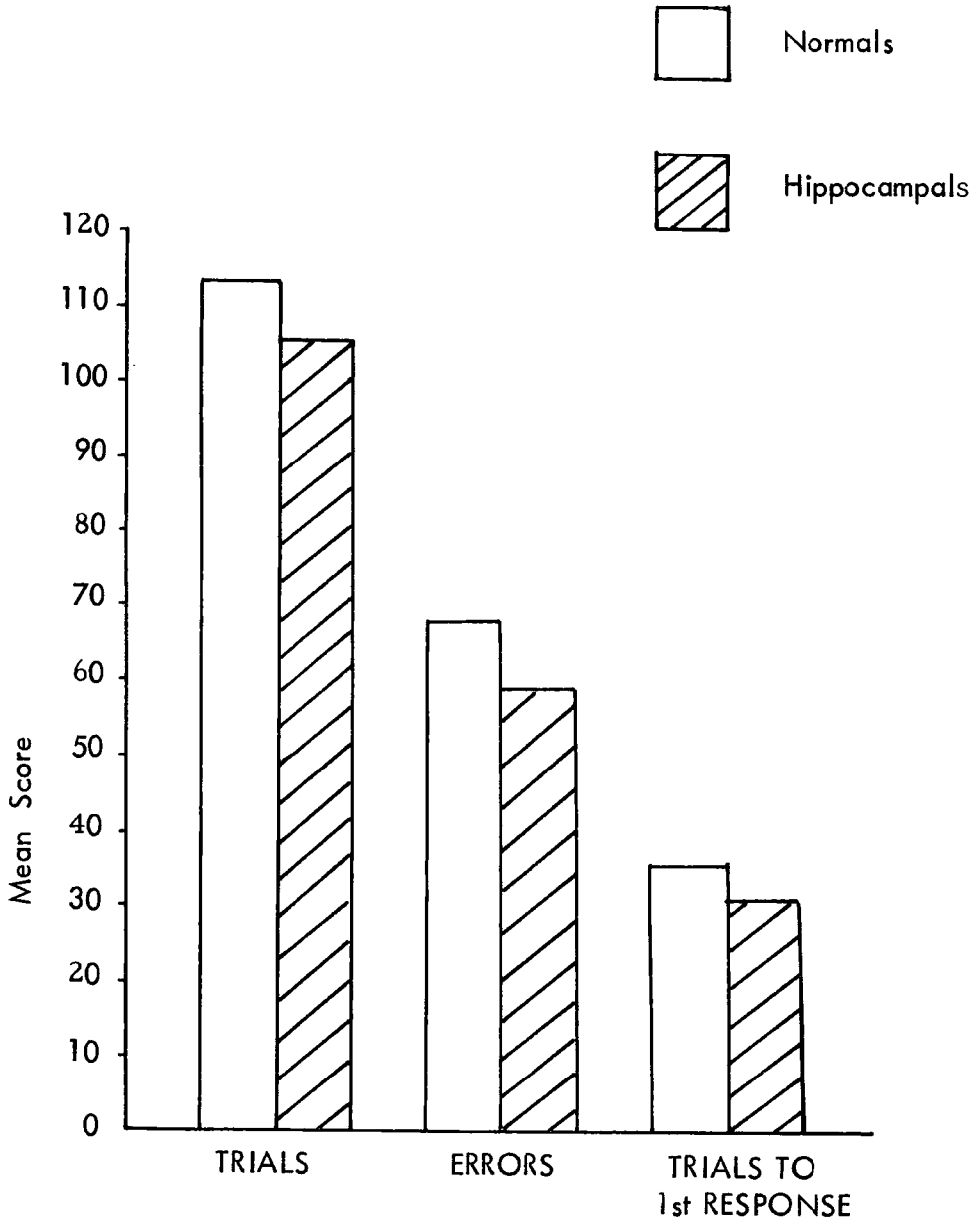


Figure 43. Histograms to show the numbers of trials, errors, and trials to the first response in autoshaping for normal and hippocampal pigeons.

therefore carried out separately for both total trials and errors to criterion, and this showed that the effect of order of training was not significant, either for trials ($F(2.12)=1.78$, $p=0.10$) or for errors ($F(2,12)=1.80$, $p=0.21$), and that none of the interactions involving the order of training factor was significant (trials, all $F's<2.42$, all $p's>0.13$; errors, all $F's<0.77$, all $p's>0.48$). There were also found to be no differences in their respective groups between those pigeons which had been trained with the horizontal bar as the positive stimulus and those trained with the vertical bar positive. In the subsequent statistical analyses, therefore, all twenty-four pigeons were treated as having completed the present experiment together, and their data were pooled appropriately.

Acquisition

Response choice

In this experiment the twelve normal and twelve hippocampal pigeons were divided into four groups of six pigeons each, and two of the groups (one normal and one hippocampal) were to be reversed immediately they reached criterion on the acquisition of the discrimination (groups NR and HR), while the other two groups (NO and HO) were to be given 500 overtraining trials prior to reversal training. The individual scores for trials and errors to criterion are presented in Tables 16 and 17 respectively, and it is clear that the two normal groups did not differ significantly on either score, and neither did the two hippocampal groups. This was confirmed using the Mann-Whitney U test (trials, $U=15$, $p>0.35$; errors, $U=16$, $p>0.41$). Therefore, in subsequent analyses of the acquisition scores, the data were pooled for the two normal subgroups and for the two hippocampal subgroups, and the mean trials and errors to criterion for the normal and hippocampal groups are summarised in Figure 44. The two groups were then compared on these two sets of scores using the Mann-Whitney U test, and no significant differences were found, either for trials or for errors ($U>42$, $p>0.10$ in each case).

Table 16

Trials to criterion in acquisition

Subjects	Normals		Hippocampals	
	Non O/T	O/T	Non O/T	O/T
1	160	150	140	190
2	130	120	160	140
3	40	30	120	120
4	100	90	130	140
5	50	110	110	60
6	130	100	120	120
Means	101.7	100.0	130.0	128.3

Table 17

Errors to criterion in acquisition

Subjects	Normals		Hippocampals	
	Non O/T	O/T	Non O/T	O/T
1	48	57	41	68
2	48	47	62	54
3	8	5	46	34
4	37	29	45	48
5	10	42	32	16
6	50	40	66	40
Means	33.5	36.7	48.7	43.3

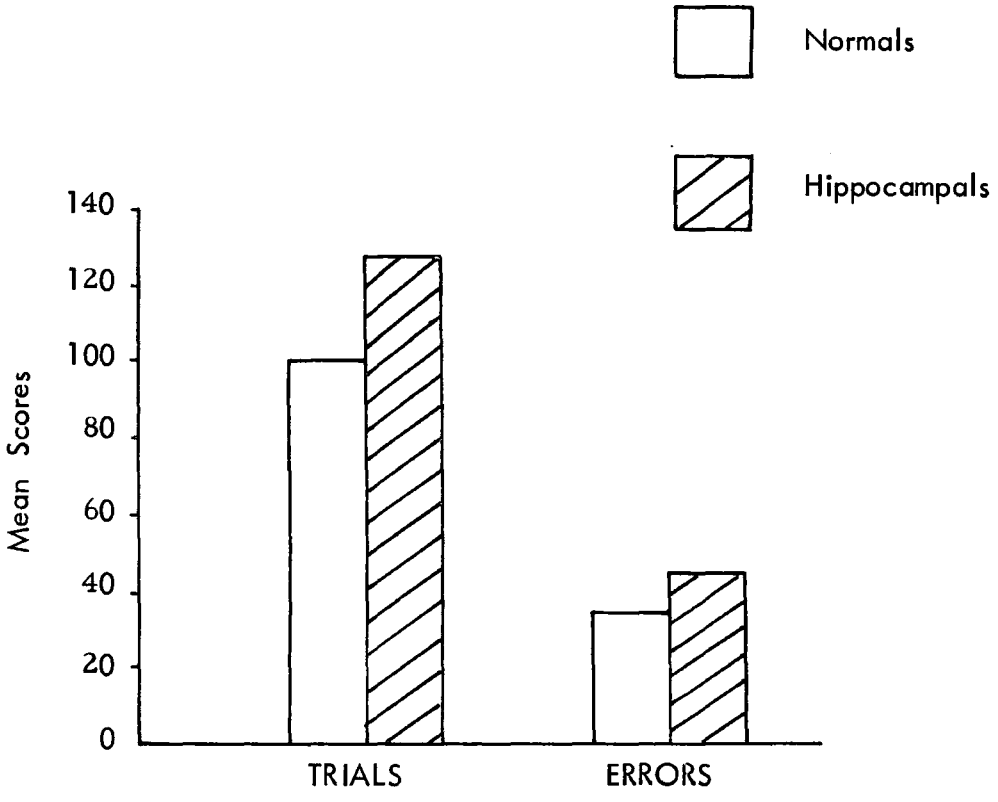


Figure 44. Mean trials and errors to criterion for normal and hippocampal pigeons in acquisition.

Response latency

In addition to the response-choice measures, response latencies were also obtained for two main purposes: first, in order to determine whether, in this experimental situation, the pigeons would show clear differences in their response latencies to the two stimuli when presented on their preferred side, particularly during the position preference stage, when response-choice measures suggest that animals are not discriminating between the positive stimulus (S+) and the negative stimulus (S-); and secondly, to determine whether the response latencies of the hippocampal pigeons differed in any way from those of the normal pigeons. In order to extract the appropriate latency measures it was first necessary to determine each pigeon's position preference. Although Macphail (1976b) suggests that this is not as straightforward a task as it would be were the subjects rats and the apparatus a Grice box, in the case of the present experiment preliminary inspection of the daily printout records revealed that the majority of the pigeons adopted a fairly rigid position habit for varying numbers of trials before they came to respond reliably to S+ regardless of the key on which it was presented. However, in order to obtain a more precise assessment of preferred side for each pigeon, a technique was used which was devised by Olton and Samuelson (1974), who were interested in comparing response-choice and response-time measures in a serial brightness reversal task in a T-maze, and who divided each reversal into five stages on the basis of the following criteria:

Stage	% Correct Responses	% Responses to Preferred Side
1. Perseveration	0	50
2. Transition	10-50	60-80
3. Position habit	40-60	90-100
4. Transition	60-80	60-80
5. Criterion	90-100	50-60

Table 18 Definition of 5 Stages in discrimination learning in terms of percentages of correct responses and of responses to the preferred position (adapted from Olton and Samuelson, 1974)

These same criteria were used here, the data being analysed in blocks of 10 trials, and it was found that only four pigeons (three normals and one hippocampal) did not pass through the Stage 3 level of performance as defined here. In each of these cases the preferred side was taken to be the side on which the majority of responses occurred in Stage 4. In the case of a fifth animal, a normal pigeon which had two blocks of trials in Stage 3, one of which showed a left key preference and the other a right key preference, the pigeon's preferred side was taken to be the key on which the majority of responses were made in the first block of trials in Stage 4. It should, perhaps, be pointed out here that in acquisition Stage 1 does not occur, because it consists of responses that continue to be made to a previously correct stimulus, and therefore is appropriately found only in reversal.

The mean latencies of correct and incorrect responses on the preferred side were then calculated separately for each of the final 8 blocks of 10 trials prior to the criterion run of 20 trials, and the mean latencies for the two groups of pigeons are presented in Figure 45. From this it can be seen that the response latencies to S+ and S- began to diverge after the fifth block prior to criterion for the normal animals, but only consistently after the third block before the criterion run for the hippocampal animals. For the normal animals t tests showed that the latency differences on the last 3 blocks of trials prior to the criterion run (Olton 1972a) were significant (block 1: $t=3.03$, $df=9$, $p<0.01$; block 2: $t=1.95$, $df=7$, $p<0.05$; block 3: $t=2.91$, $df=7$, $p<0.025$, all one-tailed), whereas, for the hippocampal animals, only the block 2 latency differences were significant ($t=2.25$, $df=7$, $p<0.05$, one-tailed).

In the experiment that Olton (1972a) carried out, normal and hippocampal rats were trained in a triangular discrimination box to discriminate between a horizontal and a vertical rectangle when presented simultaneously. He found that both groups

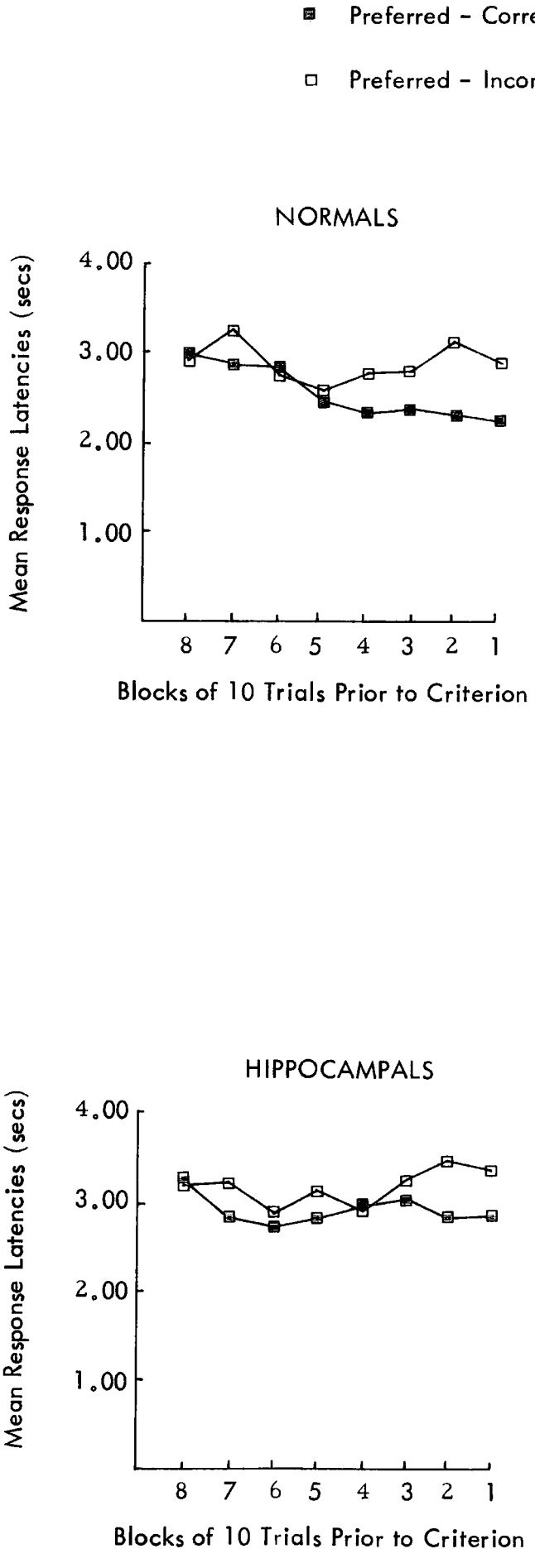


Figure 45. Response latencies to the correct and incorrect stimuli presented on the preferred key over the last eight blocks of 10 trials prior to the criterion run in acquisition.

developed consistent and significant response-latency differences on the preferred side during the acquisition of the discrimination, and also that, when position preference responses and correct choices in each block of 10 trials were averaged over animals, consistent patterns of discrimination performance emerged, which were different for normal and hippocampal rats. It therefore seemed that this type of analysis was also appropriate for the present experiment, and that it might provide some useful preliminary information concerning the discrimination performances of the two groups, prior to a more detailed analysis of the relationship between response choices and response latencies.

Consequently, in Table 19 are presented the mean numbers of correct responses and responses to the preferred side over the last 10 blocks of 10 trials, including the criterion run, for the two groups, and from this it can be seen that, for the normal pigeons, there was a sudden transition from Stage 4 to Stage 5. On the last 10 trials prior to criterion their mean number of correct responses was 7.0, and on the first block of trials at criterion they made a mean of 9.4 correct responses. The hippocampal animals made a similarly abrupt transition from Stage 4 to Stage 5, making, on average, 6.9 correct responses in the last 10 trials before criterion, and 9.7 correct responses in each of the 2 blocks of criterion trials. However, it can also be seen from this table that the hippocampal group were less consistent in their performance than the normal animals who, as a group, progressed steadily from chance levels of performance in blocks 8-5 (Stages 2-3) through Stage 4 to Stage 5, with progressive increases in their numbers of correct responses. On the other hand, the hippocampal group showed a steady increase in mean numbers of correct responses, from around chance level in blocks 8-6 to the Stage 4 level of performance in blocks 5-3, and then a sudden jump to 9.8 correct responses. (Stage 5 level of performance) on block 2, followed by a second abrupt change back to a mean of 6.9 correct responses (Stage 4) in the block of

Table 19

Mean correct responses and responses to the preferred side over the last ten blocks of 10 trials in acquisition

Blocks of 10 Trials	NORMALS Mean Responses		HIPPOCAMPALS Mean Responses	
	Correct	To Preferred Side	Correct	To Preferred Side
Criterion	9.8	5.0	9.7	4.8
Criterion	9.4	5.3	9.7	5.2
1	7.0	6.8	6.9	5.9
2	6.5	6.1	9.8	5.7
3	6.3	6.5	7.0	6.7
4	5.6	6.6	6.0	8.0
5	5.0	6.4	5.8	8.4
6	5.0	8.0	5.3	8.8
7	5.4	7.1	5.4	7.5
8	5.0	8.5	5.3	8.6

trials that immediately preceded the criterion run. Inspection of the latency data for individual pigeons suggested that this was a genuine effect rather than an artifact of the method used to obtain these mean scores, i.e., of combining together for each group data from corresponding blocks of trials which may, nevertheless, represent different stages of discrimination learning, since nine of the hippocampal pigeons showed an increase in numbers of correct responses from block 3 to block 2 prior to criterion, followed by a decrease in block 1, and finally an increase to the criterion level of performance, whereas only three of the twelve normal pigeons showed similar changes in performance from block 3 prior to criterion up to criterion itself. Nevertheless, further inspection of these data showed that individual pigeons spent different amounts of time in any particular stage in acquisition, and this had also been found, for normal rats at least, by Olton (1972a) and Olton and Samuelson (1974). However, although the pigeons, in general terms, progressed from a Stage 2, variable, level of performance, through a position preference stage (Stage 3), to criterion (Stage 5), when the stages were defined on the basis of performance on a block of 10 trials, it was found that in most cases pigeons in both groups did not progress in an entirely regular and orderly manner from Stage 2 to Stage 5. Instead, it was found that there were occasions when odd blocks of 10 trials, appropriate to a particular stage of performance, occurred in a run of blocks of 10 trials that were appropriately classified as belonging to a different stage, and that the abberant block of trials could be at a stage of performance which was either earlier or later than the run of 10 trial blocks in which it occurred. Furthermore, it appeared that this variability in behaviour occurred more frequently in the hippocampal group than in the normal group. In order to quantify this observation, therefore, each pigeon's performance in acquisition was scored in terms of the total numbers of higher stages which preceded each lower stage. These scores for individual subjects are presented in Table 20, from which it can be

Table 20

Analysis of stages of learning in acquisition in blocks of 10 trials: numbers of occasions in which higher stages preceded a lower stage of learning.

Subjects	Normals	Hippocampals
1	22	10
2	0	11
3	0	8
4	4	1
5	0	2
6	2	9
7	24	34
8	1	9
9	0	3
10	3	3
11	2	2
12	0	9
Totals	58	101

seen that the hippocampal group did, indeed, show greater variability, obtaining an overall score on this measure of 101 compared with the score of 58 obtained by the normal group. This difference was found to be significant using the Mann-Whitney U test ($U=35.5$, $p<0.05$, two-tailed). Also, five of the normal pigeons showed completely consistent progression from Stage 2 to Stage 5, as indicated by a score of 0 higher stages preceding lower stages, whereas only one of the hippocampal pigeons achieved a score of 0.

For this reason, therefore, analysing the response-choice data in blocks of 10 trials, although suitable for establishing position preferences, represented too fine a level of analysis to allow the relationship between stages of discrimination learning and changes in response latencies to be determined reliably. For this purpose it was often necessary to establish the various stages of performance on the basis of the percentages of correct responses and of responses to the preferred position in blocks of 20 or more trials. In order to be confident that the four stages into which each pigeon's acquisition performance had been divided (Stage 1 being absent in acquisition since it represents the stage at the beginning of reversal during which an animal is still responding to the former S+), this classification was carried out twice, on independent occasions, and it was found that there was complete agreement between the two assessments.

Following the classification into the four stages of performance in acquisition, the response choices in each stage were divided into four categories on the basis of the position to which the pigeon responded, and whether the response was correct or not. The four categories were, therefore:

1. Preferred-correct
2. Preferred-incorrect
3. Nonpreferred-correct
4. Nonpreferred-incorrect

Mean response latencies in each of these four categories were obtained on each of Stages 2-5 for the two groups and are presented in Figure 46. It should be noted, however, that, although data points have been plotted for all categories of response latency in all four stages, several of the points are unreliable since they represent averages of only a few responses. Specifically, very few responses were made by either group on the nonpreferred side in Stage 3, and very few incorrect responses were made to either side in Stage 5.

Where appropriate, differences in the latencies of correct and incorrect responses were analysed by t test, and the one-tailed test was used for latency differences on the preferred side in Stages 3 and 4, since it was predicted that preferred-correct latencies would be shorter than preferred-incorrect latencies; the two-tailed test was used for all other response latency differences.

Normal pigeons

Preferred-key responses

During Stage 2 the correct latency tended to be shorter than the incorrect latency, although these differences were not significant ($t = 0.63$, $df = 18$, $p > 0.50$). From Stage 2 to Stage 3 both correct and incorrect latencies on the preferred key showed a decrease, which was more rapid for the correct responses so that, in Stage 3, the correct latency was noticeably shorter than the incorrect latency. This difference, which was found to be significant ($t = 1.82$, $df = 34$, $p < 0.05$), continued into Stage 4, and again was found to be significant ($t = 2.43$, $df = 27$, $p < 0.025$). In Stage 5, the correct latency showed a further slight decrease but, as stated above, because so few incorrect responses were made in this stage, latency differences were not analysed.

Nonpreferred-key responses

Response latencies on the nonpreferred key showed a different trend. In Stage 2 there was very little difference between correct and incorrect latencies (means: 2.88 secs

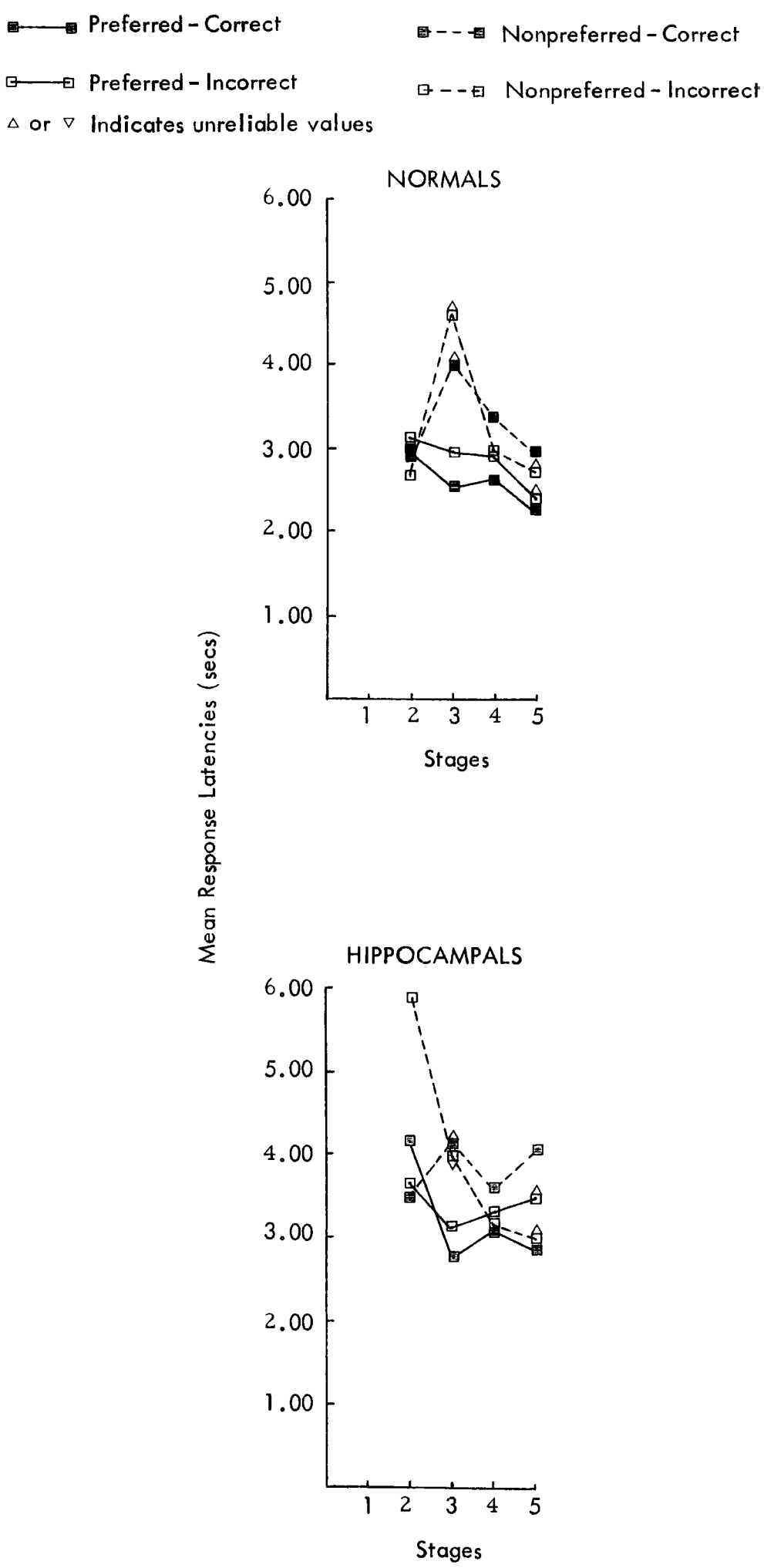


Figure 46. Response latencies to the correct and incorrect stimuli on preferred and nonpreferred keys in each of the four stages in acquisition.

and 2.70 secs, respectively). From Stage 2 to Stage 4 both correct and incorrect latencies increased to some extent, this change being more marked for the correct latency so that, in Stage 4, it was longer than the incorrect latency, although these differences were not significant ($t=0.75$, $df=25$, $p>0.40$). Finally, in Stage 5, the nonpreferred-correct latency became shorter, although it was still longer than the preferred-correct latency, and this difference was found to be significant ($t=3.40$, $df=21$, $p<0.01$).

Hippocampal pigeons

Preferred-key responses

In Stage 2 the correct response latency was slightly longer than the incorrect latency, but this difference was not significant ($t=2.06$, $df=22$, $p>0.05$). From Stage 2 to Stage 3 both latencies became shorter and crossed over so that, in Stage 3, the correct latency was now shorter than the incorrect latency, and this difference was found to be significant ($t=1.99$, $df=44$, $p<0.05$). The two response latencies then increased together into Stage 4 but the difference between them was not significant ($t=0.41$, $df=48$, $p>0.6$). In Stage 5 the correct latency then showed a further slight decrease (from 3.11 to 2.85 secs).

Nonpreferred-key responses:

Again, the nonpreferred-key responses behaved differently. In Stage 2 it appears that the correct latency was considerably shorter than the incorrect latency, but this difference was not significant ($t=2.08$, $df=13$, $p>0.05$). primarily because several fairly long latencies made by two of the animals contributed to the mean incorrect latency of 5.90 secs. From Stage 2 to Stage 4 the correct latency increases slightly, while the incorrect latency decreased considerably, so that the two crossed over and the incorrect latency was shorter than the correct latency in Stage 4. This was found to be significant ($t=2.35$, $df=29$, $p<0.05$, two-tailed). Finally, from Stage 4 to

Stage 5 the nonpreferred-correct latency showed a further increase, and it was found to be significantly longer than the preferred-correct response latency in Stage 5 ($t = 2.97$, $df = 23$, $p < 0.01$).

In summary, therefore, the traditional response-choice measures of trials and errors to criterion indicated that there were no significant differences between the two groups in the acquisition of this discrimination. However, when analysed in terms of response latencies to the correct and incorrect stimuli on the preferred side, it was found that the normal pigeons began to respond significantly faster to the correct stimulus than they did to the incorrect stimulus somewhat earlier than the hippocampal pigeons. Also, when acquisition performance was divided into stages of learning, evidence was obtained for greater variability in the performance of the hippocampal pigeons. They did not show the same degree of continuity as was shown by the normal pigeons in progressing from the early, somewhat variable, level of performance that defines Stage 2, through the adoption of a position habit in Stage 3, and the breaking of this position habit in Stage 4, to the criterion level of performance in Stage 5.

Further analysis of the response latency data, categorised for correct and incorrect responses on the preferred and nonpreferred keys, showed that both groups of pigeons began to discriminate between S+ and S- in terms of response latencies while they were still responding predominantly to position (Stage 3). However, the latency differences for the normal group were more marked than those for the hippocampal group. Also, for the normal group this clear difference in latencies between correct and incorrect responses on the preferred side was maintained into Stage 4, whereas this was not so for the hippocampal group, their correct and incorrect response latencies on the preferred side in Stage 4 being not significantly different. Furthermore, in Stage 5 the preferred-correct latencies were significantly faster than the nonpreferred-correct latencies for both groups, showing that, although the pigeons had given up their

position habits (Stage 3) and in Stage 5 were responding consistently to the relevant cue, they still retained a preference for one key or position, a finding which would not have been detected by a response-choice measure.

Reversal

Response choice

The total numbers of trials and errors to criterion in reversal for the individual pigeons are presented in Tables 21 and 22 respectively, and the mean trials and errors for each of the four groups are summarised in Figure 47. By comparing the scores in Tables 21 and 22 with those in Tables 16 and 17 respectively, it can be seen that each of the four groups of pigeons took more trials and made more errors in reversal than in acquisition, as expected, although it should be noted that one of the non-overtrained hippocampal pigeons showed the opposite effect. To facilitate comparison between the acquisition and reversal scores for the four groups, the mean trials and mean errors in acquisition have been included in Figure 47. In order to evaluate these comparisons a three-factor analysis of variance with repeated measures, the factors being lesion treatment x overtraining x acquisition/reversal, was carried out on the data for trials and for errors. Overall, the main effect of the lesion treatment was found to be not significant, both for trials ($F(1,10)<0.03$, $p>0.84$) and for errors ($F(1,10)=0.30$, $p=0.60$). Similarly, no significant effect was found for overtraining on trials to criterion ($F(1,10)=0.24$, $p=0.64$) or on errors ($F(1,10)=0.30$, $p=0.60$). However, the analysis confirmed that the effects of reversal training were highly significant, for both trials ($F(1,10)=84.18$, $p<0.00005$) and for errors ($F(1,10) = 146.06$, $p< 0.00005$) and that the interaction between lesion treatment and reversal training was also significant (trials, $F(1,10)=8.73$, $p=0.014$; errors, $F(1,10)=4.91$, $p=0.049$). Finally, the lesion treatment x overtraining x reversal training interaction was found to be not significant, either for trials ($F(1,10)=0.65$, $p= 0.44$) or for errors

Table 21

Trials to criterion in reversal

Subjects	Normals		Hippocampals	
	Non O/T	O/T	Non O/T	O/T
1	220	300	240	300
2	190	240	290	160
3	180	210	150	190
4	310	280	160	250
5	120	290	250	90
6	250	180	80	190
Means	211.7	250.0	195.0	196.7

Table 22

Errors to criterion in reversal

Subjects	Normals		Hippocampals	
	Non O/T	O/T	Non O/T	O/T
1	141	190	120	179
2	110	123	187	84
3	91	153	94	119
4	170	152	88	151
5	73	177	155	54
6	152	94	40	107
Means	122.8	148.2	114.0	115.7

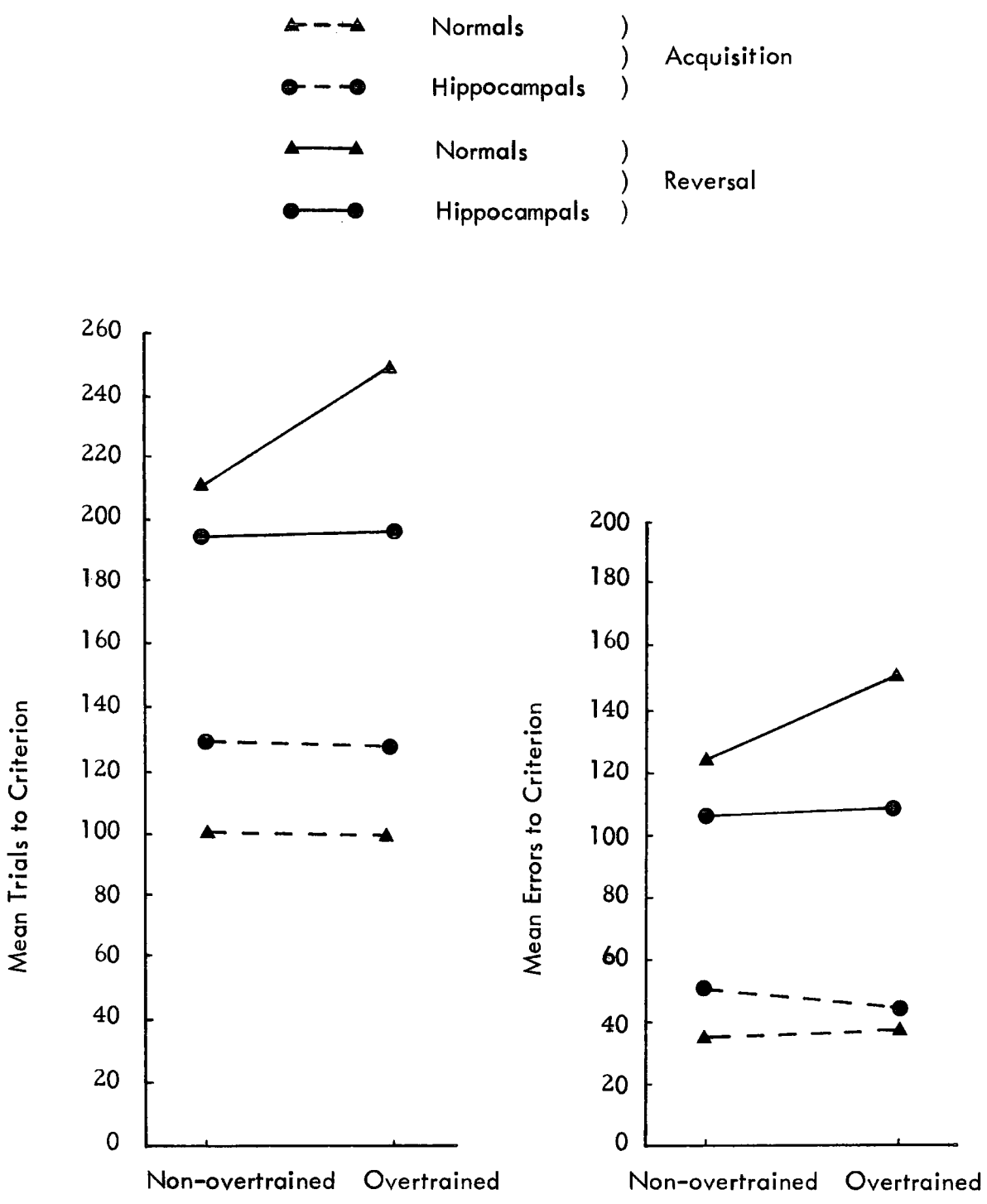


Figure 47. Mean trials and errors to criterion in acquisition and reversal for non-overtrained and overtrained normal and hippocampal pigeons.

($F(1,10)=0.19$, $p=0.68$). Separate two-factor analyses were also carried out on the reversal data alone, but neither main effect of lesion treatment or overtraining was found to be significant, nor was the interaction between them, both for trials and for errors (with 1 and 20 df, all F's <1.65, all p's >0.21).

Comparison of the acquisition and reversal scores of the normal and hippocampal pigeons in Figure 47 reveals that, for both trials and errors, the order between the two main groups reversed from acquisition to reversal. Thus, the increase in the numbers of trials and of errors from acquisition to reversal was greater for the normal groups than it was for the hippocampal groups, and this was reflected in the significant lesion x reversal interaction effect noted above. To examine this effect further, therefore, a measure referred to as a reversal index was determined for each pigeon by subtracting the number of trials to criterion in acquisition from the number of trials in reversal. The reversal indices for the individual pigeons, and the means for the four groups, are presented in Table 23. A two-factor analysis of variance carried out on these scores confirmed that there was a significant lesion effect ($F(1,20)=8.21$, $p=0.009$), but no significant effect was found for overtraining ($F(1,20)=0.96$, $p=0.34$), and the lesion treatment x overtraining interaction was also found to be not significant ($F(1,10)=0.69$, $p=0.42$).

From this analysis it can be seen that, although the differences between the hippocampal and the normal pigeons in reversal were not statistically significant, the hippocampal pigeons tended to take fewer trials and make fewer errors to criterion in reversal than the normal pigeons. Also, while overtraining had no effect on the performance of the hippocampal pigeons, it appears to have increased the number of trials and the number of errors made to criterion by the normal pigeons. Furthermore, a significant lesion treatment x acquisition/reversal training effect was obtained, and by analysing the reversal index, a measure which reflected the increase in the number

Table 23

Reversal index for each subject

Subjects	Normals		Hippocampals	
	Non O/T Trials	O/T Trials	Non O/T Trials	O/T Trials
1	60	150	100	110
2	60	120	130	20
3	140	180	30	70
4	210	190	30	110
5	70	180	140	30
6	120	80	-40	70
Means	110.0	150.0	65.0	68.3

of trials in reversal over those in acquisition, it was found that the hippocampal pigeons showed a significantly smaller increase compared with the normal pigeons. This clearly shows, therefore, that the hippocampal pigeons were not impaired on the reversal task, and perhaps even suggests the possibility that their reversal performance was facilitated.

Several further measures of reversal performance were obtained from the data from the individual trials for each pigeon in an attempt to determine the nature of the differences between the groups, and in particular, the effects of overtraining on the reversal performance of the normal pigeons. The first of these measures, the number of errors prior to the first correct response, was obtained as a measure of perseverative behaviour, in the same way that it was in the serial spatial reversals task (see Chapter 4). These data for the individual pigeons are presented in Table 24, from which it can be seen that the hippocampal pigeons tended to show less perseverative responding to the former S+ than the normal pigeons, although a two-factor analysis of variance showed that neither of the main effects, nor the interaction between them, was significant (lesion treatment, $F(1,20)=3.01$, $p=0.09$; overtraining, $F(1,20)=0.09$, $p=0.76$; lesion treatment x overtraining, $F(1,20)=0.05$, $p=0.93$). The second measure, errors to equal choice (Matyniak and Stettner, 1970), is related to the first measure, but provides a more comprehensive assessment of the ability of the various groups to extinguish responses to the former S+. This measure was obtained by calculating the total number of errors in reversal prior to the first block of 10 trials in which five or more correct responses occurred, and the individual scores and group means are presented in Table 25. From the mean values for each group it can be seen that, in both the normal and the hippocampal pigeons, overtraining tended to increase the numbers of errors to equal choice, and that this effect was greater for the normal pigeons. However, a two-way analysis of variance again revealed that neither lesion treatment nor

Table 24

Errors to 1st correct response in reversal

Subjects	Normals		Hippocampals	
	Non O/T	O/T	Non O/T	O/T
1	82	65	3	19
2	37	31	21	38
3	23	57	53	40
4	39	7	38	34
5	30	91	47	15
6	66	11	19	17
Means	46.2	43.7	30.2	27.2

Table 25

Equal choice errors in reversal

Subjects	Normal		Hippocampals	
	Non O/T	O/T	Non O/T	O/T
1	116	137	34	139
2	60	66	118	64
3	47	127	58	79
4	72	81	58	79
5	58	97	97	42
6	105	53	32	57
Means	76.3	93.5	66.2	76.7

Table 26

Position habit responses in reversal

	Normals		Hippocampals	
	Non O/T	O/T	Non O/T	O/T
Mean No. of 90% position response blocks	8.0	7.3	4.7	6.7
Mean % position preference responses	71.8	69.3	66.3	67.2
Mean No. position hypotheses	108.3	115.8	85.7	93.8

overtraining was significant, and also that the interaction between these two factors was not significant (all F's<1.10, all p's>0.30, with 1 and 20 df).

The other three measures were obtained from the raw data in order to provide information about the position responses of each of the four groups of pigeons, and they are identical to the measures that Silveira and Kimble (1968) derived from their data on the reversal performance of hippocampal rats on a brightness task. The scores obtained by each group on these three measures are presented in Table 26. The first of these, the mean number of 90% position response blocks, refers to the numbers of blocks of 10 trials in which each pigeon made 9 or more responses to one key; the second value, mean % position preference responses, is the number of responses made to the preferred key during reversal, expressed as a percentage of the total trials to criterion; and for the third measure, a position hypothesis was defined as a sequence of responses to the animal's preferred position which included responses to both S+ and S-, and all the responses contained in such sequences were totalled for each pigeon. From these scores, it is clear that overtaining had little effect on the position responding of the normal pigeons, or of the hippocampal pigeons, although there was a slight increase in the mean number of blocks of 10 trials in which 9 or more position responses occurred in the overtrained hippocampal group when compared with the other hippocampal group. However, since the three scores obtained by the overtrained hippocampal group are very similar to those obtained by the two normal groups, it would seem to be more appropriate to consider any apparent differences in the scores between the two hippocampal groups as being due to the slightly lower tendency of the nonovertrained hippocampal pigeons to adopt a position hypothesis. Nevertheless, a two-factor analysis of variance that was carried out on these various scores showed that none of the differences between the four groups was significant (all F's<1.2, all p's>0.25, with 1 and 20 df).

In summary, although none of the differences between the groups on the perseveration, resistance to extinction, and position responding measures was significant, it appears that the hippocampal pigeons tended to make fewer perseverative responses to the former S+ at the beginning of reversal, that their resistance to extinction was slightly lower, and that they tended to make fewer position habit responses than the normal pigeons. Furthermore, although it was found that overtraining appeared to retard the reversal learning of the normal group, from the data presented in Tables 25 and 26, it can be seen that the pigeons in the overtrained group showed a small increase in resistance to extinction, but this was accompanied by only a minor increase in position responding.

Response latency

Preliminary, as well as more detailed analyses of the relationship between response latency and response choice, similar to those undertaken for acquisition, were carried out on the reversal data. For these purposes each pigeon's position preference in reversal, and the latencies of correct and incorrect responses, were obtained from the raw data using the same procedures that were used for the acquisition data.

As before, an initial analysis was made of the mean response latencies of correct and incorrect responses on the preferred side on the final 8 blocks of 10 trials prior to the criterion run of 20 trials, and these data for each of the four groups are presented in Figure 48. For the nonovertrained normal pigeons, it can be seen that the correct and incorrect response latencies began to separate consistently on the sixth block prior to criterion. As before, correlated t tests (one-tailed in all cases) on the preferred-correct and preferred-incorrect response latencies on each of the final 3 blocks revealed that the correct latencies were significantly shorter than the incorrect latencies (block 1, $t = 2.29$, $df = 5$, $p < 0.05$; block 2, $t = 5.24$, $df = 4$, $p < 0.01$; block 3, $t = 2.85$, $df = 4$, $p < 0.025$). The response latencies of the overtrained

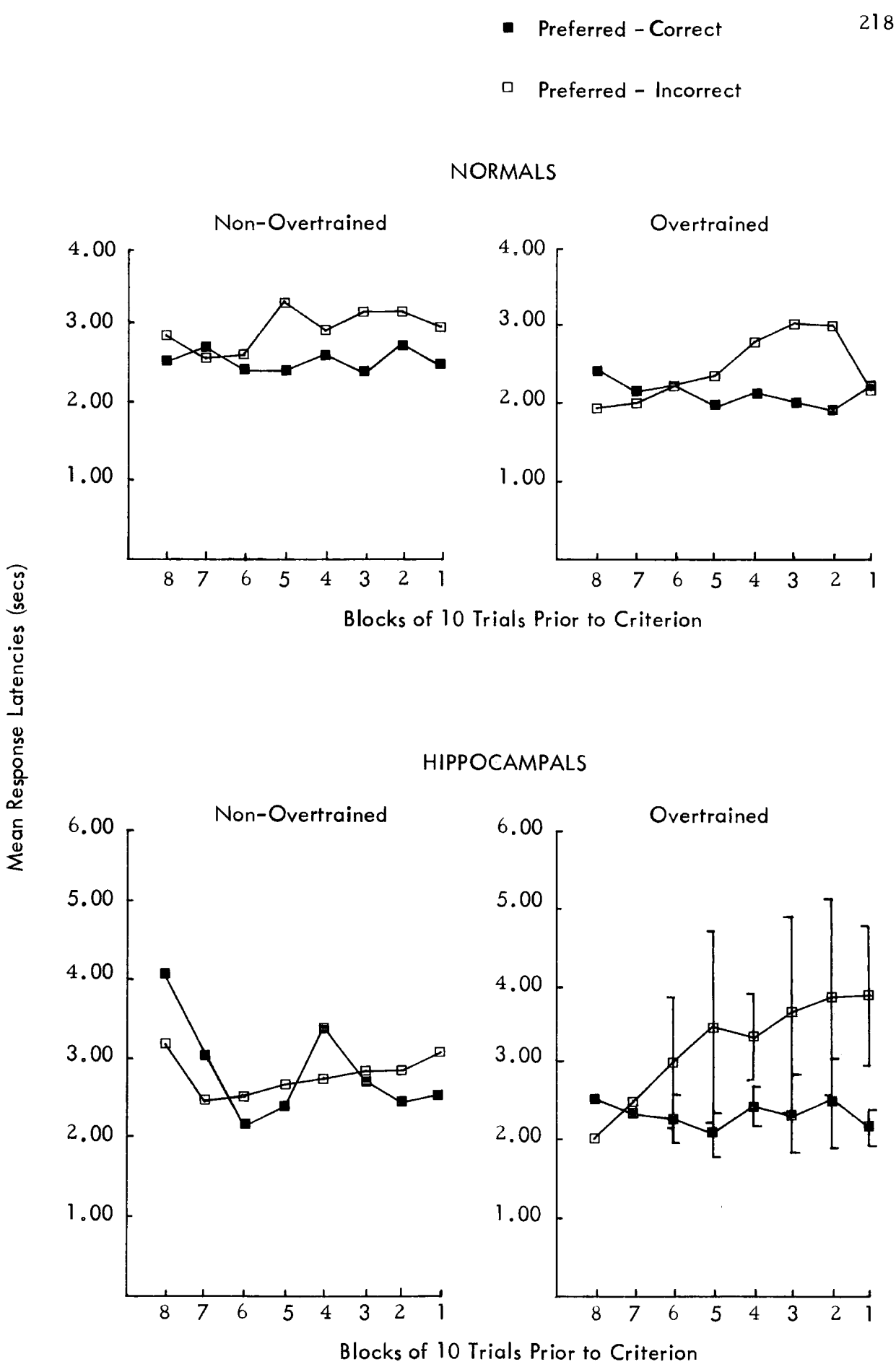


Figure 48. Response latencies to the correct and incorrect stimuli presented on the preferred key over the last eight blocks of 10 trials prior to the criterion run in reversal.

normal pigeons clearly began to diverge by the fifth block, and this trend continued until block 2, but finally the correct and incorrect latencies converged in the block immediately preceding the criterion run. Although the response latencies in blocks 2 and 3 appear to be widely separated, because of the considerable variability in the latency scores of the pigeons in this group, only the latency differences in block 2 were significant ($t = 3.54$, $df = 4$, $p < 0.025$).

In contrast, it is clear that the correct and incorrect response latency differences of the non-overtrained hippocampal group were rather erratic, shorter preferred-correct latencies first occurring on block 6 and continuing in block 5, but then not occurring again until block 2. When analysed by t tests, it was found that none of the differences on the final 3 blocks prior to criterion was significant. Finally, it appears that the response latencies of the overtrained hippocampal pigeons began to diverge reliably as early as the seventh block and that this trend continued up to and including the block immediately preceding the start of the criterion run. However, there was considerable variability in the latency scores in this group, as indicated by the standard deviations shown on this graph, and the mean incorrect latencies on each block of 10 trials tended to be distorted by some relatively long response latencies made by two or three pigeons in each block, the particular animals varying from block to block, and consequently the t tests carried out on the final three blocks revealed that only the block immediately preceding the first criterion block contained correct response latencies that were significantly shorter than the incorrect latencies ($t = 2.46$, $df = 4$, $p < 0.05$). It is, perhaps, noteworthy that over the 8 blocks of trials that were considered for each group, the preferred-correct response latencies for three of the groups, the exception being the non-overtrained hippocampal group, tended to remain relatively constant, and that any separation between these and the preferred-incorrect response latencies was due to increases in the latter.

A further preliminary analysis was carried out to compare the discrimination reversal performance of each of the four groups over the final 10 blocks of 10 trials. In Table 27 the mean numbers of correct responses and responses to the preferred side on each of the last 10 blocks of trials, including the criterion run, are presented for each group. From this it can be seen that, as in acquisition (see Table 19) both normal groups and both hippocampal groups made fairly abrupt transitions from a Stage 4 level of performance in the last block of trials prior to criterion to the Stage 5 level of performance, and also that, on the whole, all four groups showed a steady progression from levels of performance appropriate to Stage 2 in block 8 up to Stage 5. Nevertheless, some slight fluctuations in performance between blocks 4 and 2 were noted for all four groups. Also, it was clear that, although individual 10-trial blocks for each pigeon had been classified into five stages on the basis of the percentages of correct responses and of position preference responses (see Table 18) the group data presented in Table 27 were not always readily classified. This suggested therefore that, as in acquisition, corresponding blocks of 10 trials from the various pigeons in a group again did not always represent the same stage of discrimination learning. Consequently, it seemed appropriate to carry out the same type of analysis of the stages of performance in reversal as was done for acquisition. Again, it was found that, in general terms, all pigeons progressed from the Stage 1, perseverative, level of performance through a position preference stage (Stage 3) to criterion levels of performance (Stage 5). But, as expected, since it had already been found in the acquisition phase of this experiment, and had been reported for normal rats by Olton (1972a) and also by Olton and Samuelson (1974), confirmation was obtained that varying numbers of trials in reversal were spent in each of the five stages by the various pigeons. It was also found, as in acquisition, that the hippocampal pigeons showed a greater tendency towards irregular progression from Stage 1 to Stage 5, compared with the normal pigeons, when the data were

Table 27

Mean correct responses and responses to the preferred side over the last ten blocks of 10 trials in reversal

NORMALS

Blocks of 10 Trials	Non-overtrained		Overtrained	
	Mean Responses		Mean Responses	
	Correct	To Preferred Side	Correct	To Preferred Side
Criterion	9.8	4.8	9.8	5.2
Criterion	9.5	5.2	9.7	5.0
1	6.3	6.7	7.3	5.7
2	6.7	7.0	6.3	6.0
3	7.3	6.7	6.7	6.3
4	5.3	8.3	6.2	7.2
5	4.5	8.8	6.3	8.0
6	4.5	9.5	5.0	8.0
7	4.7	9.7	5.0	8.7
8	4.0	8.3	4.5	8.2

HIPPOCAMPALS

Blocks of 10 Trials	Non-Overtrained		Overtrained	
	Mean Responses		Mean Responses	
	Correct	To Preferred Side	Correct	To Preferred Side
Criterion	9.5	4.5	9.3	5.0
Criterion	9.8	4.8	9.2	4.8
1	6.7	5.7	6.7	6.0
2	6.5	7.5	6.7	7.0
3	4.7	6.0	5.3	5.7
4	5.0	7.3	6.0	5.0
5	4.5	6.8	5.0	7.3
6	4.8	7.5	4.5	9.2
7	4.8	8.2	3.8	8.5
8	4.2	6.8	4.4	8.2

analysed in blocks of 10 trials.

Using the same technique as before, the numbers of stages that were out of order in each pigeon's reversal performance were obtained and are presented for each of the four groups in Table 28. From this it can be seen that there was relatively little difference between the scores of the two normal groups, although the nonovertrained group showed a tendency towards slightly higher scores, and the overtrained hippocampal group, but that the nonovertrained hippocampal group obtained a score that was noticeably larger than any of the other three scores. However, it can also be seen that this larger score was contributed to mainly by only two of the pigeons in this group, and a two-way analysis of variance confirmed that neither the two main effects nor the interaction was significant ($F(1,20)<2.64$, $p>0.11$ in each case).

Once again, in order to analyse the relationships between stages of performance, response choice, and response latencies in reversal learning, it was clear that a more reliable determination of the stages of learning was required, and that it would be obtained by considering the numbers of correct responses and of position preference responses in blocks of 20 trials or more, rather than in 10 trial blocks. Thus, the procedure used for the acquisition data was again used here.

The mean response latencies in the four categories, preferred-correct, preferred-incorrect, nonpreferred-correct, and nonpreferred-incorrect, were calculated for each of the five stages for the two normal groups and the two hippocampal groups, and are presented in Figure 49. As before, a number of the data points for each group, indicated on the figure, must necessarily be regarded with some caution since they represent mean latency scores obtained from inadequate numbers of responses. However, it is perhaps noteworthy that in some of these cases, and particularly the preferred- and nonpreferred-incorrect response latencies in Stage 5, there is a good degree of correspondence across all four groups, suggesting that the small numbers of response latency scores obtained in

Table 28

Analysis of stages of learning in blocks of 10 trials in reversal : numbers of occasions on which higher stages preceded a lower stage of performance

Subjects	Normals		Hippocampals	
	Non O/T	O/T	Non O/T	O/T
1	2	1	19	9
2	2	3	26	2
3	1	6	3	1
4	12	4	2	0
5	6	4	12	1
6	14	4	0	12
Totals	37	22	62	25

these two categories by each group were, nevertheless, representative.

Where appropriate, the response latency differences on the preferred-side in Stages 3 and 4 were analysed by one-tailed t tests, since it was predicted that preferred-correct latencies would be shorter than preferred-incorrect latencies. All other latency differences were analysed using two-tailed tests.

In Stage 1, by definition, no correct responses were made on either key, and therefore no statistical analysis was carried out on the response latencies in this stage. Very few nonpreferred-incorrect responses were made in Stage 2 by the non-overtrained normal pigeons and the overtrained hippocampal pigeons, the latter group also making few nonpreferred-correct responses. Finally, in Stage 5, very few incorrect responses were made on either key by all pigeons.

Normal pigeons

Non-overtrained group

Preferred-key responses

During Stage 2 the pigeons tended to respond more rapidly to the incorrect stimulus on the preferred side, although the latency differences were found not to be significant ($t = 1.61$, $df = 21$, $p > 0.10$). From Stage 2 to Stage 3, although both correct and incorrect latencies decreased, the reduction in the former was greater so that, in Stage 3, there was no difference between the two (mean correct latency, 2.55 secs; mean incorrect latency, 2.54 secs). While the correct latency continued to decrease from Stage 3 to Stage 4, the incorrect latency now increased and was found to be significantly longer than the correct latency ($t = 3.09$, $df = 11$, $p < 0.01$). Finally, in Stage 5, the correct response latency increased again, and the data available suggest that the incorrect latency showed a similar, if not greater, increase.

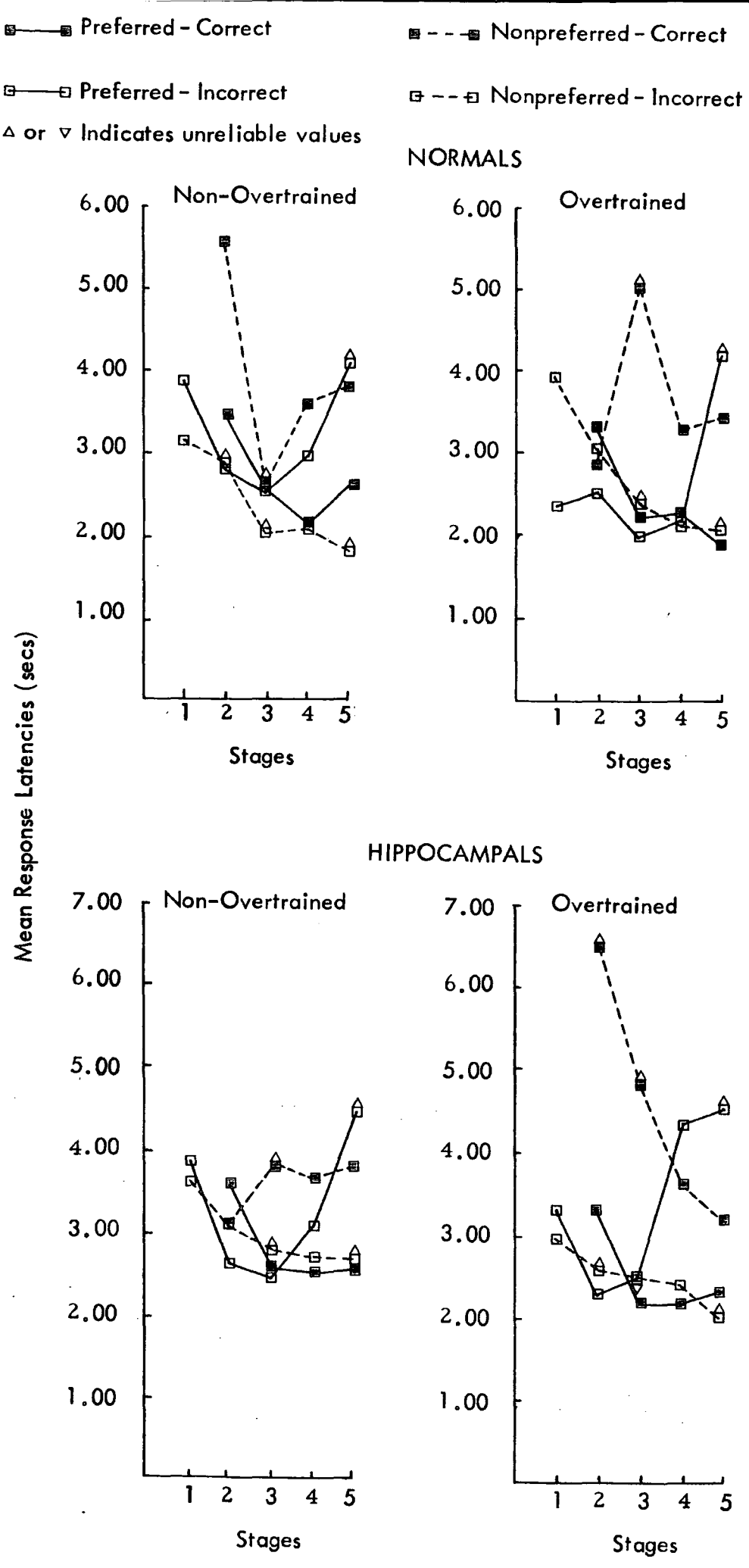


Figure 49. Response latencies to the correct and incorrect stimuli on preferred and nonpreferred keys in each of the five stages in reversal.

Nonpreferred-key responses

Response latencies on the nonpreferred key showed a somewhat different trend. In Stage 2 the correct latency was usually fairly long (mean = 5.51 secs), and anyway was rather longer than any of the other three categories. From Stage 2 to Stage 4 both correct and incorrect latencies showed an overall decrease, and in Stage 4 the difference between the two was found to be significant ($t = 5.63$, $df = 7$, $p < 0.01$), the incorrect latency being the shorter of the two. In Stage 5 the nonpreferred correct latency lengthened slightly, and it was found to be significantly longer than the preferred correct latency ($t = 3.27$, $df = 11$, $p < 0.01$).

Overtrained group

Preferred-key responses

The preferred-correct latency in Stage 2 was longer than the incorrect latency, and the difference between them was significant ($t = 2.44$, $df = 34$, $p < 0.05$). From Stage 2 to Stage 3 both categories of response latency were reduced, the correct latency decreasing more than the incorrect latency, and the difference between them was found to be not significant ($t = 1.27$, $df = 42$, $p > 0.10$). In Stage 4 both the correct and the incorrect response latencies increased slightly to approximately the same value, (mean correct latency, 2.30 secs; mean incorrect latency, 2.24 secs), but in Stage 5 the correct latency again became shorter while the incorrect latency, according to the limited data available, increased somewhat.

Nonpreferred-key responses

In comparison, the response latencies on the nonpreferred key in Stage 2 were very similar (means: correct latency, 2.94 secs; incorrect latency, 3.12 secs). From Stage 2 to Stage 4 the correct latency showed an increase, while the incorrect latency showed a marked decrease, and the difference between the two latencies was found to be significant ($t = 5.43$, $df = 17$, $p < 0.01$). In Stage 5 the nonpreferred-correct latency

increased again slightly and was significantly longer than the preferred-correct response latency ($t = 4.53$, $df = 11$, $p < 0.001$).

Hippocampal pigeons

Non-overtrained group

Preferred-key responses

The correct response latency was significantly longer than the incorrect latency in Stage 2 ($t = 3.11$, $df = 34$, $p < 0.01$), but both subsequently decreased in such a way that by Stage 3 the correct latency (mean, 2.56 secs) was only marginally longer than the incorrect latency (mean, 2.43 secs). From Stage 3 to Stage 4 a marked change in the pattern of the latency differences occurred, with the correct latency remaining fairly constant while the incorrect latency increased somewhat so that it was now significantly longer than the correct latency ($t = 2.60$, $df = 14$, $p < 0.025$). Finally, in Stage 5 the correct latency showed virtually no change from the previous stage while the incorrect latency showed a tendency towards being fairly long.

Nonpreferred-key responses

The correct and incorrect response latencies in Stage 2 were almost identical (means, 3.14 secs, and 3.13 secs, respectively), but diverged from Stage 2 to Stage 4, when the mean correct latency became noticeably longer than the mean incorrect latency, although because of the variability in the scores, both within and between subjects, the difference was not significant ($t = 1.37$, $df = 10$, $p > 0.20$). In Stage 5 the nonpreferred-correct latency increased slightly and it was found to be significantly longer than the preferred-correct latency ($t = 4.66$, $df = 11$, $p < 0.001$).

Overtrained group

Preferred-key responses

In Stage 2 the correct response latency was significantly longer than the incorrect latency ($t = 4.43$, $df = 21$, $p < 0.001$), but subsequently the two response latencies

crossed over so that, in Stage 3, the correct latency was shorter than the incorrect latency, although the difference between the two was not significant ($t = 0.80$, $df = 37$, $p > 0.40$). From Stage 3 to Stage 4 the correct latency remained constant, but the incorrect latency increased considerably and was significantly longer than the correct latency in Stage 4 ($t = 2.83$, $df = 11$, $p < 0.01$). In Stage 5 the correct latency increased very slightly, and so, apparently, did the incorrect latency.

Nonpreferred-key responses

Insufficient numbers of responses were made on the nonpreferred key in Stages 2 and 3, and therefore the mean response latencies obtained from these data should be regarded as unreliable. However, when compared with the other three groups, it is clear that the mean nonpreferred-incorrect latency of the overtrained hippocampal group was of approximately the same order of magnitude in Stage 2 and these incorrect latencies showed a similar trend from Stage 1 to Stage 5 as in the other groups. In Stage 4 the correct latency was significantly longer than the incorrect latency ($t = 2.23$, $df = 11$, $p < 0.05$), and in Stage 5 the nonpreferred-correct latency showed a small decrease, but was still significantly longer than the preferred-correct latency ($t = 4.77$, $df = 11$, $p < 0.001$).

In summary, although there were found to be no significant differences between the normal and hippocampal pigeons in either trials or errors to criterion in reversal, and no significant overall effect of overtraining, there was the suggestion that overtraining tended to increase the numbers of trials taken and errors made by the normal pigeons, but had no effect on the reversal performance of the hippocampal pigeons. Also, when reversal scores were compared with the scores in acquisition it was found that the hippocampal pigeons took relatively fewer trials to criterion in reversal, and therefore effectively showed superior reversal performance compared with the normal pigeons. When the response latencies of correct and incorrect responses on the preferred key were compared for the four groups (i.e., NR, NO, HR, and HO - see p. 185) reliably shorter latencies to the correct stimulus over the final 3 blocks of 10 trials

prior to criterion were found only for the NR group. Significantly shorter correct response latencies were only found to occur on the second block before the criterion run for the NO group, and on the block of trials immediately preceding the start of the criterion performance for the HO group. None of the differences between the correct and incorrect response latencies of the HR group was found to be significant. However, in reversal, the hippocampal pigeons did not show the same degree of variability in performance over Stages 1-5 that they showed in acquisition.

The results of the detailed analysis of the latencies of responses to the correct and incorrect stimuli on the preferred and nonpreferred sides in each of the five stages in reversal are necessarily complicated, although a number of observations may be made from them. First, there appear to be considerable similarities between the four groups when response latencies in corresponding stages are compared, and secondly, there appear to be several important differences between the acquisition and the reversal data. In acquisition it was found that there were no significant differences between the correct and the incorrect response latencies on the preferred side in Stage 2 in either group. Then, in Stage 3, both normal and hippocampal pigeons showed reliably faster response latencies to the correct stimulus when it was presented on the preferred side, an effect which was maintained in Stage 4 by the normal group but not by the hippocampal group. However, in reversal, the incorrect response latency on the preferred side in Stage 2 for each group was noticeably faster than the correct response latency, an effect which indicates that, although they had given up responding consistently to the former S+, and were beginning to adopt a position habit, they maintained their preference for the former S+. It then appears that this effect was continued into Stage 3, the position habit stage, since the preferred-correct and the preferred-incorrect latencies in this stage in reversal were not significantly different for any of the groups. Such a difference first appeared in Stage 4 for three of the four

groups, the exception being group NO. However, as in acquisition, the preferred-correct latencies in Stage 5 were significantly faster than the nonpreferred-correct latencies for all groups, showing again a distinct position preference while the pigeons were responding consistently to the visual cue.

Discussion

The main findings from this experiment showed that the hippocampal pigeons were not impaired on either the acquisition or the reversal of a simultaneous visual discrimination, and that no significant effects of overtraining were found in either main group, although overtraining had a rather greater tendency to retard reversal learning in the normal pigeons, due to an increased resistance to extinction, than it did in the hippocampal pigeons. Evidence was obtained which showed that the hippocampal pigeons were significantly more variable in their progression from Stage 2 to Stage 5 in acquisition, compared with the normal pigeons, and although not significant, there was also a strong tendency for the hippocampal pigeons to show greater variability in progressing from Stage 1 to Stage 5 in reversal, indicating that the response strategies used by the hippocampal pigeons were different from those used by the normal pigeons. Support for this came from a detailed analysis of the individual responses during reversal, which revealed that the hippocampal pigeons responded less to spatial cues than the normal pigeons. This was further supported by the finding that the hippocampal pigeons took longer to develop response latency differences to the two stimuli when they were presented on the preferred side than the normal pigeons did, and did not maintain them for as long. However, more detailed analysis of the response latencies to the correct and incorrect stimuli on the preferred and nonpreferred sides in each of the four (acquisition) or five (reversal) stages revealed very similar performances by the various groups.

The finding that hippocampal lesions in pigeons do not impair their ability to

acquire a simultaneous visual discrimination has also been obtained in hippocampal mammals by numerous investigators (e.g., rats: Silveira and Kimble, 1968; Winocur and Salzen, 1968; cats: Teitelbaum, 1964; Webster and Voneida, 1964; Nonneman and Isaacson, 1973; monkeys: Schram, 1970; Mahut, 1971, 1972), although there have been a few exceptions (e.g., Olton, 1972a; Woodruff and Isaacson, 1972). Nevertheless, it perhaps should be noted that the hippocampal group did take more trials and make more errors in acquisition than the normal group. More important, however, is the finding that the hippocampal pigeons appeared to be using different strategies, compared with the normal pigeons, who were found to develop response latency differences to the correct and incorrect stimuli when presented on their preferred side earlier in acquisition than the hippocampal pigeons did, and also to maintain them for longer. Other evidence for behavioural differences between the two groups was obtained from the detailed analysis of the stages of discrimination learning that had been proposed by Olton (1972) and Olton and Samuelson (1974), and which showed in the present experiment that the hippocampal pigeons did not progress from the earlier, variable stage, through the position preference stage, to the final stage in which criterion performance is achieved in as consistent and sequential a manner as the normal pigeons did. This suggests that the hippocampal pigeons were not always responding to the visual and/or spatial cues as reliably as the normal pigeons were.

The finding that the hippocampal pigeons did not take more trials or make more errors to criterion than the normal pigeons during reversal, and therefore were also not impaired on the reversal of a nonspatial discrimination, is similar to that obtained in hippocampal rats (Samuels, 1972), cats (Isaacson et al, 1968), and monkeys (Mahut, 1971; Jones and Mishkin, 1972; Mahut and Zola, 1973). However, when the reversal performance of the normal and hippocampal pigeons was compared with their performance during acquisition, it was found that, effectively, the hippo-

campal pigeons were superior to the normal pigeons. Although it is tempting to compare this effect with the facilitated visual reversal performance in monkeys with hippocampal lesions that was reported by Schram (1970) and by Zola and Mahut (1973), in both cases the hippocampal monkeys were superior to the normal animals in both acquisition and reversal, and therefore these findings are not strictly comparable with those of the present experiment. Nevertheless, this relative superiority of the hippocampal pigeons emphasises their lack of impairment on this reversal task.

Although no significant effects of lesion treatment or of overtraining were found, and the interaction between them was also not significant, it would appear that the results of this experiment are in the predicted direction. In the hippocampal pigeons there were no differences in the numbers of trials and errors to criterion between the nonovertrained and the overtrained groups, showing, therefore, that overtraining had not affected the reversal performance of these animals. On the other hand it was found that overtraining tended to retard reversal learning in the normal pigeons. However, a more detailed analysis of the individual data in reversal revealed that overtraining in the normal group did not affect their responding to the former S+, nor did it have a particularly noticeable effect on their position responding, although the reports of Mackintosh (1965) and Matyniak and Stettner (1970) were to some extent confirmed by the finding in the present experiment that overtraining tended to increase resistance to extinction in the normal pigeons, as shown by a small (22.5%) increase in their errors to equal choice. The finding of a minimal effect of the overtraining trials on position responding suggests that the horizontal-vertical discrimination used in this experiment was not as easy a task as the preliminary tests had suggested, since it has been found that overtraining retards reversal learning in birds on easy visual tasks, has no effect on more difficult tasks, and produces an ORE on extremely difficult visual discriminations (Sutherland and Mackintosh, 1971, pp. 437-438). It would seem,

therefore, that the increase in the number of trials and errors to criterion in the overtrained normal group, compared with the nonovertrained normal group, is accounted for by the increased resistance to extinction that they showed.

The detailed data analysis for the hippocampal pigeons showed that they made, if anything, fewer perseverative responses to the previously correct stimulus at the beginning of reversal compared with either normal group, and in the majority of studies it has been found that hippocampal mammals have equally little difficulty in giving up their responses to the former S+ during reversal training on a nonspatial task (O'Keefe and Nadel, 1978, p. 283). Compared with the nonovertrained hippocampal group, the overtrained hippocampal pigeons also tended to show a slight, but insignificant, increase (15.9%) in their resistance to extinction. Furthermore, their scores on this measure (equal choice errors in reversal) are, in fact, lower than those obtained by the respective normal groups, and this can be taken as additional evidence to support the previous findings (Chapters 3 and 4) that hippocampal lesions in pigeons, as in mammals (Schmaltz and Isaacson, 1967; Nonneman et al, 1974), are not necessarily impaired in extinction, and that extinction deficits, when they do occur, are more likely to be task-dependent (see Chapter 5). The position response analysis is of interest, since it shows that, although the several scores obtained by the overtrained hippocampal group are generally of the same order of magnitude as those obtained by both normal groups, they nevertheless are all lower than the respective normal scores, suggesting a slight tendency towards fewer responses to position. But in particular, although again not significantly different, the nonovertrained hippocampal group had markedly fewer position blocks and made fewer position hypothesis responses than either normal group, supporting the proposal made earlier that the hippocampal pigeons appear to make less use of, or respond less consistently to, spatial cues compared with normal pigeons.

Whereas the measures of the variability of performance in acquisition were significantly different, the hippocampal pigeons obtaining a rather higher score than the normal pigeons, in reversal none of the differences between the groups was significant. Nevertheless, there was still a tendency for the hippocampal pigeons towards more variability, as indicated by higher scores on this measure, than the corresponding normal groups. Also present was the suggestion that overtraining tended to reduce this variability in both groups, and perhaps more so in the hippocampal animals. Finally, the response latency data showed that only the nonovertrained normal group developed reliably different response latencies to S+ and S- during the final 3 blocks of trials before criterion. However, when the response latencies were considered in relation to the several stages of learning that occur in discrimination tasks, it was found that the four groups were very similar in each of the four categories of response latency. Moreover, in Stage 2, the latencies of responses to the incorrect stimulus on the preferred side were, in all four groups, significantly faster than the preferred-correct response latencies, showing that, although the pigeons had all given up responding to the former S+ and were beginning to adopt a position habit, they were still showing a marked preference for the previously correct stimulus. In Stage 5 the opposite effect was found: all four groups showed significantly faster response latencies to the correct stimulus when it was presented on their preferred side than when it was presented on their nonpreferred side, this time demonstrating that they were showing a marked preference for one position, or key, while responding reliably to the visual stimulus. These results clearly show, therefore, that the hippocampal pigeons were as able as the normal pigeons to attend to one cue while responding to the other, thereby showing normal selective attention. Thus, the present results again do not support the proposal that hippocampal animals are impaired in their ability to inhibit attention (Douglas, 1967; Kimble, 1968; Silveira and Kimble, 1968). This further supports

findings obtained in the colour probability experiment (Chapter 3 - see pp. 122-124). Furthermore, the results of the analysis of the individual trials in reversal showed that the hippocampal pigeons did not make more errors prior to the first correct response, and did not show increased resistance to extinction, as shown by the errors to equal choice scores. These results, therefore, do not support the response-inhibition or response-perseveration hypotheses of hippocampal dysfunction that have been proposed by Kimble and Kimble (1965), McCleary (1966), Uretsky and McCleary (1969), and more recently, Altman et al (1973). The overall lack of impairment on both the acquisition and the reversal of this visual discrimination, together with the response-latency data, show that the hippocampal pigeons were not suffering from an inability to shift responses, as proposed by Olton (1972a), a finding which was also obtained from the serial position reversal task reported in Chapter 4. The present results therefore provide further evidence to support the proposal that the effects of hippocampal lesions in pigeons are different from those produced by hyperstriatal lesions, since Macphail (1971, 1975a, 1976a, 1976b) has argued that the hyperstriatal region is involved in either a response-inhibition or a response-shift mechanism. However, it is suggested instead that the present data indicate that the hippocampal pigeons to some extent responded abnormally to spatial cues, and that this could be due to an impaired ability to use spatial hypotheses. Several proposals have been made implicating the hippocampus in the regulation of hypotheses, although the suggestions of Silveira and Kimble (1968) and Kimble and Kimble (1970) rely on fixated hypothesis behaviour in hippocampal animals being due to impaired selective attentional processes. But as noted above, the hippocampal pigeons in the present experiment showed normal attentional mechanisms. An alternative version of the hypothesis regulation hypothesis has been proposed by Pribram et al (1969), in which they regard the hippocampus and the amygdala as being part of a functional system in which the hippocampus is involved in modifying hypotheses on

the basis of disconfirming events (e.g., nonreinforcement) and the amygdala is concerned in maintaining hypotheses in the light of confirming events (i.e., successful outcomes). It can be seen, therefore, that this is little more than a slightly modified version of the selective attention model that was proposed earlier (Douglas and Pribram, 1966) in which the hippocampus was said to be responsible for the switching of attention from stimuli that were associated with nonreinforcement and the amygdala was concerned with maintaining attention to those stimuli that were associated with reward. It is clear that these hypotheses are also inappropriate to the present data.

More recently, it has been suggested that the amnesic syndrome seen in humans with hippocampal damage is due to interference effects (Weiskrantz and Warrington, 1975), and further, that it may be possible to explain the behavioural changes that follow hippocampal damage in animals in similar terms, which would have the added benefit of reconciling the human and animal data, which, until recently, have appeared to disagree much more than they agree. Winocur (1979) devised an experiment to test the hypothesis that the hippocampus in animals is involved in the control of interference by presenting rats with high or low interference tasks during the acquisition or retention of a visual pattern discrimination, and found that the hippocampal rats were more impaired than the normal rats by the high interference tasks. He suggests, therefore, that an appropriate interpretation of the increased response perseveration that has been found to occur in hippocampal rats on certain tasks following prior training on an incompatible task is that it is due to interference effects as a result of a deficit in the processing of available information. Thus "in situations where hippocampal animals are unable to dissociate contextual conditions and adjust their strategies in accordance with changes in the task, their tendency often is to persist with the most recently established response" (Winocur, 1979, p. 344). In the present experiment, however, the hippocampal pigeons were not impaired on the reversal of the visual discrimination following previous training

on what Winocur would necessarily regard as an incompatible task, namely the acquisition of the discrimination, and thus they did not show interference effects. A further task on which hippocampal animals also ought to be impaired due to interference effects is extinction, but it was found in two of the previous experiments (Chapters 3 and 4), as well as in the present task, that the hippocampal pigeons did not show an extinction deficit. Indeed, the only situation in which an extinction deficit was found in the hippocampal pigeons was that following DRL 10 training, a task which could be regarded as being compatible with extinction.

On the other hand, the spatial information processing model (O'Keefe and Nadel, 1978) proposes that, without the ability to use efficient place learning strategies, animals with hippocampal lesions have instead to use less efficient strategies involving external stimulus-response associations which, it is argued, are very prone to interference effects from similar situations. Winocur (1979) argued that his results could only be explained in terms of this model if it was assumed that the hippocampal and normal rats used different response strategies in order to learn the discrimination, but no differences between the strategies of the two groups were found. However, in the present experiment, as noted above, evidence was obtained which suggested that the hippocampal pigeons were using different response strategies, both in acquisition and in reversal, and that they appeared to make less use of spatial hypotheses than the normal pigeons. It is proposed, therefore, that the present results are not in agreement with the explanation offered by Winocur (1979), but that they are, instead, consistent with the spatial information processing theory of hippocampal function.

CHAPTER 7 Acquisition of a Delayed Spatial Alternation Task

Introduction

The discovery by Scoville (1954) and others (see the Introduction, p. 3), of a severe memory defect in man following the bilateral surgical removal of part of the temporal lobe and hippocampus suggested to a number of investigators that animals with hippocampal lesions should be impaired on delayed response tasks. Thus, Mishkin (1954) and Orbach et al (1960) both found that monkeys with bilateral resection of the temporal lobe and hippocampus performed poorly on the acquisition of a delayed response task. Similar effects were later reported in hippocampal cats (Karmos and Grastyan, 1962; Ungher and Sirian, 1970 - both cited in O'Keefe and Nadel, 1978, pp. 326-327) and rats (Niki, 1962). However, Mahut and Cordeau (1963) found that two hippocampal monkeys trained in a WGTA were not impaired on a delayed response task, although one of them was impaired when trained on a delayed spatial alternation task. This suggested that an important feature of the second task was not the delay, but the spatial factor. Subsequently, Racine and Kimble (1965) found that hippocampal rats trained in a T-maze were impaired on the postoperative retention of a delayed spatial alternation task, and others have reported impaired acquisition of the task, also in a maze (e.g., Greene, 1971; Means, Leander, and Isaacson, 1971). In addition, there have been reports of delayed spatial alternation deficits in monkeys (Rosvold, Mishkin, and Swarcbart, 1964 - cited in Rosvold and Swarcbart, 1964) and rats (Niki, 1966; Riddell, Malinchoc, and Reimers, 1973) trained in an operant chamber.

Support for the notion that spatial factors were more important than the delay factor was provided by an experiment by Mahut (1971), who reported that a small group of hippocampal monkeys trained in a WGTA were superior to normal monkeys on delayed response and nonspatial (go, no-go) delayed alternation tasks, but were impaired on a delayed spatial alternation task. However, similar deficits were also

found in monkeys with ablations of either frontal or temporal cortex, or the amygdala, and furthermore, although two of the hippocampal monkeys failed the task completely, the other two were able to learn it, but at a reduced rate. On the other hand, Brown et al (1969) found that cats trained in a modified WGTA were not impaired on a delayed spatial alternation problem. A similar result was obtained by Waxler and Rosvold (1970), who trained hippocampal monkeys in a WGTA and found that, as a group, they were not impaired on a delayed spatial alternation task, although from the individual data that are presented, it is clear that four of the hippocampal monkeys made no more errors than any of the eight normal monkeys, while the other four made many more errors. In explaining these results, Waxler and Rosvold suggested that one of the important factors responsible for the different levels of performance may have been the use of different response strategies by the various hippocampal monkeys, More recently, Stevens and Cowey (1972, 1973) have reported that rats with hippocampal lesions were not impaired when trained in a two-lever operant chamber on a spatial alternation task similar to that used by Niki (1966). Although the rats in each of these three experiments had also been trained in a previous experiment, it would appear that potentially the most disruptive of these, because it was a task which was the most incompatible with a spatial alternation problem, was the position reversal experiment carried out by Niki. Similarly, Riddell et al (1973), who had also reported a hippocampal deficit in spatial alternation in an operant chamber, had previously trained their animals on a position discrimination. Evidence has already been presented which suggests the importance of the effects of prior training on the subsequent performance by hippocampal animals on various tasks (e.g., extinction, DRL performance, and passive avoidance learning - see Chapters 1 and 5). Further evidence that hippocampal mammals are not impaired in the performance of a sequential task in an operant chamber comes from an experiment by Jackson and Strong (1969), who found that hippocampal

rats were, in fact, superior to normal rats in learning to press two levers in various alternating sequences, and subsequently in learning sequences involving three levers.

The present experiment was therefore designed to investigate the effects of hippocampal lesions in pigeons, first on the acquisition of a nondelayed spatial alternation in an operant chamber, and subsequently on their performance on essentially the same task, but with various delays inserted between trials.

Method

Subjects

Twenty-four pigeons were used, twelve of which had received bilateral hippocampal lesions, the remaining twelve pigeons being either sham-operated or unoperated controls. All twenty-four pigeons had been trained in two other experiments, the reversal of a visual form discrimination and the delayed colour alternation task. The three experiments were run in a balanced design, and details of this, and of the pretraining procedures used have been presented in Chapter 5 (see pp. 183-185).

Apparatus

A standard two-key Campden Instruments operant chamber was used, which was lit by a white houselight, and both keys could be illuminated with white light.

Procedure

Nondelayed spatial alternation training

At the beginning of each session the houselight was on and both keys were lit. Completing FR5 on either key switched off both keylights, during which 3 secs access to food provided reinforcement if the five responses had been on the correct key, whereas incorrect responses switched off the houselight for 3 secs TO. Responses on the two keys were counted separately, so that whether or not the trial was correct depended on which key first accumulated five responses. At the end of either reinforcement or TO there was a very brief (0.2 secs) delay interval during which the predetermined counters used

to control the individual FR5 schedules were automatically reset. The keylights then came on again to signal the start of the next trial. On the first trial of each day the correct key was varied according to a Gellerman sequence, and on subsequent trials the correct key was the one opposite that on which FR5 had been completed on the previous trial, regardless of whether or not the trial had been correct. Each pigeon was given 50 trials a day for 40 days.

Delayed spatial alternation training

Delayed alternation training began on day 41. The procedure used here was essentially the same as that used in the nondelayed task, except that the delay interval was varied daily according to a predetermined random schedule and was either 1, 2, 3, 5, or 10 secs long. In addition, the pigeons were now given correction trials in which the position of the correct key only switched to the opposite side following a correct trial. Furthermore, each pigeon was run each day until it had completed 100 reinforced trials, and training continued for 25 days. Each pigeon therefore received 5 days of training on each of the five delays.

In both stages of the experiment electromechanical counters were used to record the daily scores of correct and incorrect trials, together with the numbers of trials on which FR5 was completed on left and right keys.

Results

Histology

The histological reconstructions of the lesions of the twelve hippocampal pigeons used here are presented in Chapter 6.

Nondelayed spatial alternation

The data for the mean percentages of correct trials were analysed in blocks of 5 daily sessions over the 40 days of training. Since the pigeons in this experiment were divided into three subgroups, B1, B2, and B3, and two of these groups were run

in either one or both of the other experiments prior to being trained in the present experiment, the order in which the groups were trained was treated as a separate factor in the analysis of variance to determine whether it had had any effect on performance in the present task.

A three-factor repeated measures analysis of variance revealed that, with the scores collapsed over the 8 blocks of 5 days, there was no significant difference between the normal and the hippocampal groups on the nondelayed spatial alternation task ($F(1,6) < 0.005$, $p > 0.90$), but there was a significant effect over blocks of 5 days ($F(7,42) = 69.54$, $p < 0.00005$), although the groups x blocks interaction was not significant ($F(7,42) = 0.48$, $p = 0.85$). However, with the normal and hippocampal pigeons combined in each case there was a significant effect of order of training on the mean percent correct trials collapsed over blocks of 5 days ($F(2,12) = 4.01$, $p = 0.046$). Multiple comparisons using the Tukey test (Keppel, 1973, p. 138) were therefore carried out on the mean scores contributing to this main effect, and it was found that the only significant difference was that between the B1 and B3 subgroups ($p < 0.05$, 1 and 12 df), the differences between B1 and B2 and between B2 and B3 being considerably smaller than the critical value at the 0.05 level of significance. These data for each of the three subgroups are summarised separately for the normal and hippocampal groups of pigeons in Figure 50, and from these three graphs it can be seen that the changes in performance that occurred over the 8 blocks of 5 days are very similar, both within and between subgroups B1, B2, and B3, but that the overall levels of performance show a general improvement from B1 to B3. The analysis of variance, however, showed that neither the lesion treatment x order of training interaction ($F(2,12) = 1.53$, $p = 0.25$), nor the order of training x blocks of days interaction ($F(14,84) = 1.71$, $p = 0.07$), nor the lesion treatment x order of training x blocks of days interaction was significant ($F(14,84) = 0.92$, $p = 0.54$).

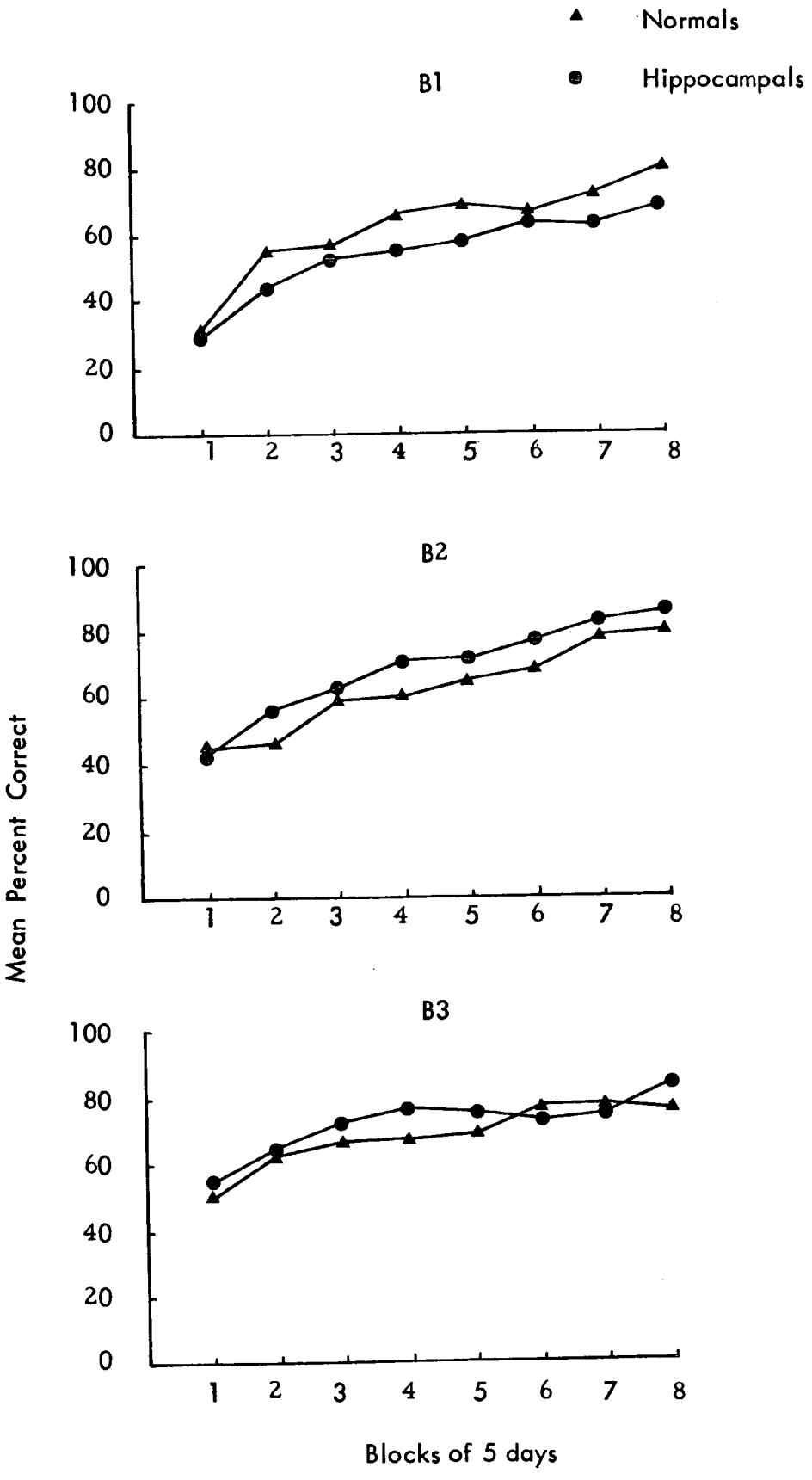


Figure 50. Performance of normal and hippocampal pigeons according to their order of training on the nondelayed spatial alternation task.

It would seem, therefore, that the effect of prior training on one or both of the other experiments in this series was equivalent to a general practice effect, since the significant order of training effect was found only when the data from the normal and the hippocampal groups were pooled, and none of the interactions involving the order factor was significant. Furthermore, separate two-factor analyses of variance on the data from each of the B1, B2, and B3 subgroups confirmed that there were no significant differences between the normal and hippocampal groups, with the data collapsed over blocks of 5 days ($F(1,6)<1.85$, $p>0.22$ in each case), and the interaction between lesion treatment and blocks of days was not significant ($F(39, 234)<1.05$, $p>0.39$ in each case). However, further inspection of Figure 50 reveals that the relationship between the performances of the normal and the hippocampal animals reversed from B1 and B2. Therefore, in order to examine in more detail the changes that occurred from B1 to B2 to B3 separately for the normal and hippocampal pigeons, the data for these two main groups were replotted and are presented in Figure 51. From the shape of the learning curves alone, it now appears that the order of training did affect the normal and the hippocampal pigeons differently, and that it had a greater effect on the performance of the hippocampal pigeons on subsequent tasks than it did on the normal pigeons. However, despite the apparent separation between the data for hippocampal groups B1 and B2 over the whole of the 8 blocks of days, and between B2 and B3 over at least the first half of training, when the degree of variability of the individual scores in each of the three subgroups is indicated by the addition of the standard deviations in each case (mean $\pm$ 1 s.d.), the overlap in the scores is immediately apparent.

By comparison, the learning curves for the B1, B2, and B3 subgroups of normal pigeons show considerable overlap over the whole of the training period, except for the first block of 5 days, and the addition of any measures of variability would possibly

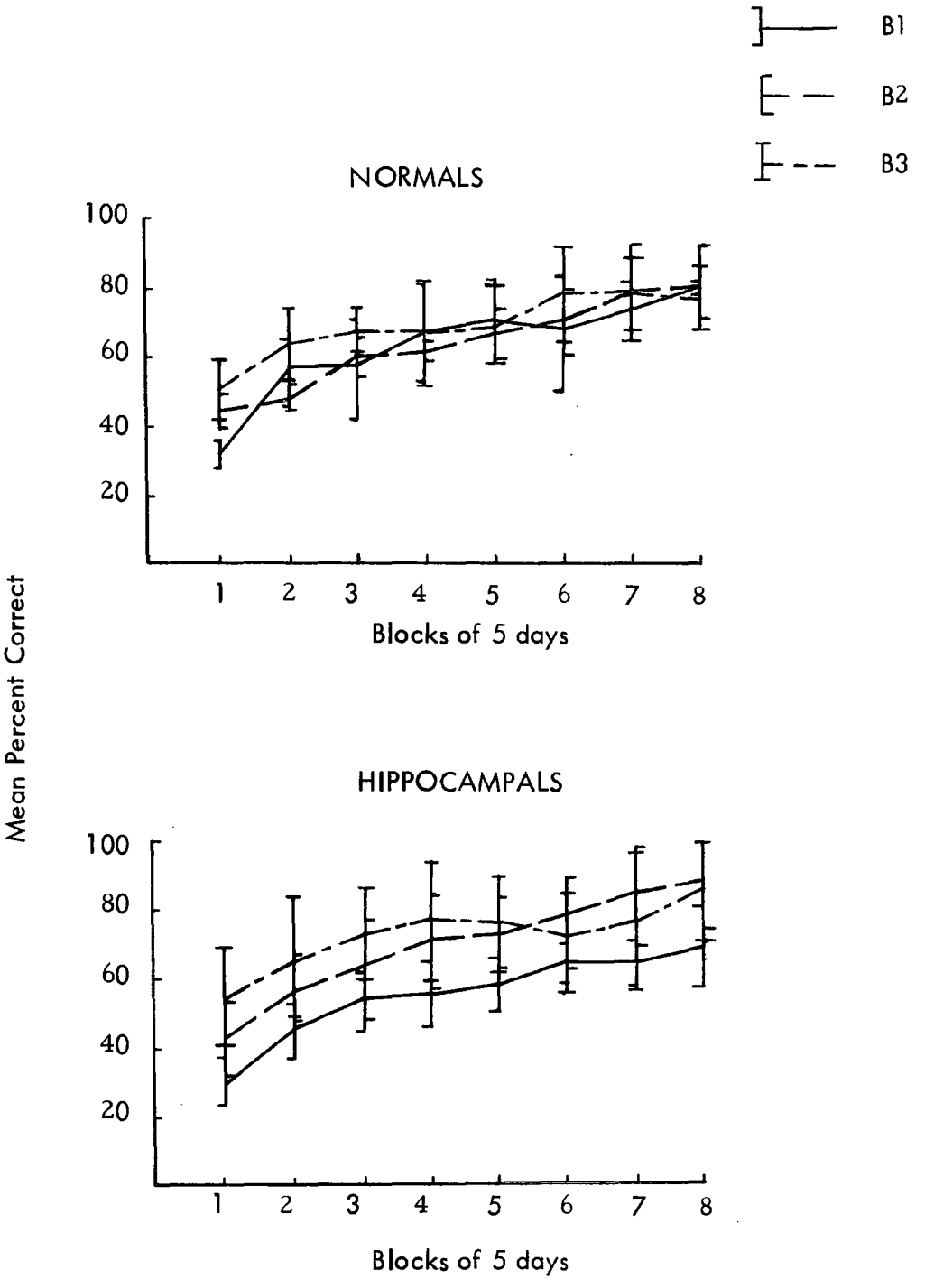


Figure 51. Comparisons of performance of the 3 normal groups and the 3 hippocampal groups to show the effects of training on performance in nondelayed spatial alternation task (data in Figure 50 redrawn).

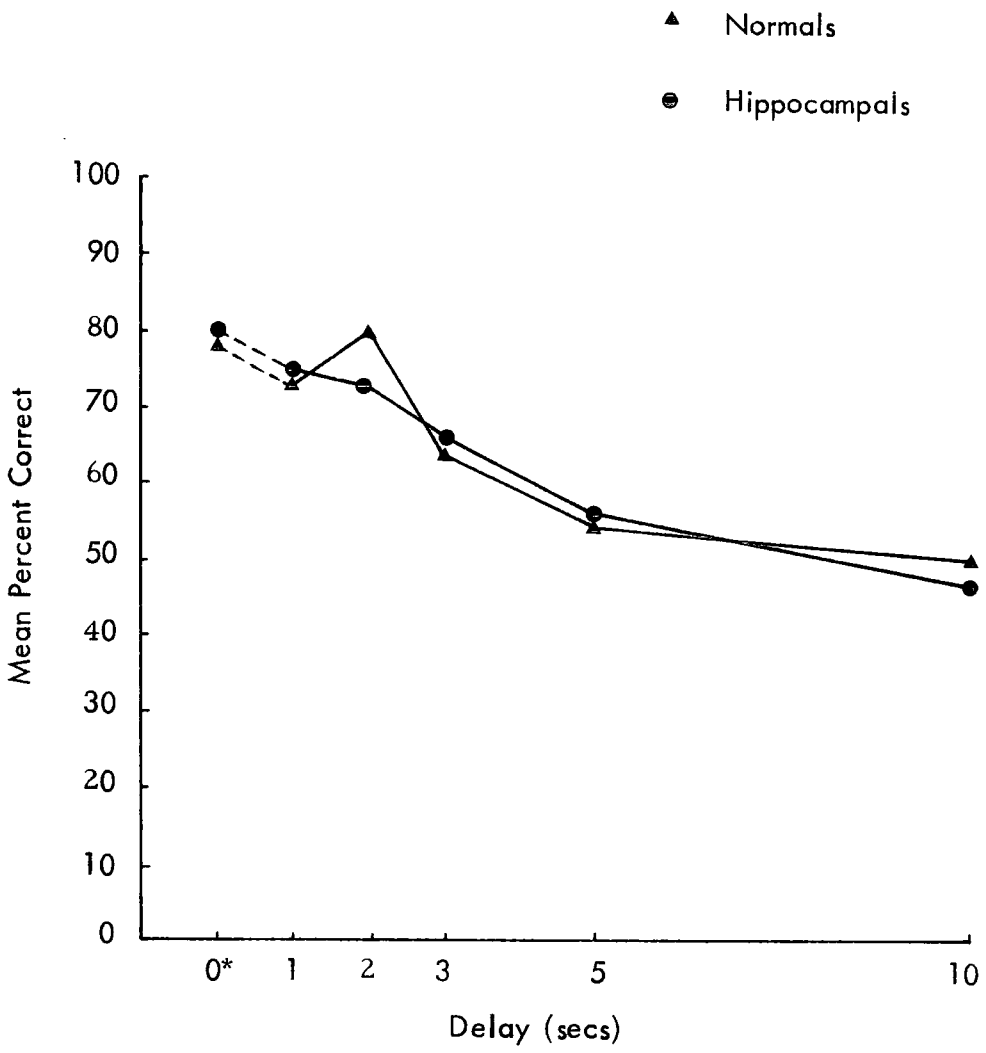
seem superfluous. Nonetheless, standard deviations have been plotted in the same manner as for the hippocampal groups, and, as expected, they confirm the earlier observations, although it should, perhaps, be noted, that there is clearly no overlap in the scores for groups B1 and B2 on the first block of 5 days. This further analysis therefore, confirms the earlier findings and supports the notion that, in this experiment, training on previous tasks provided a general practice effect which affected the normal and the hippocampal pigeons similarly.

In summary, both normal and hippocampal groups of pigeons learned the nondelayed alternation task equally well, although, even after 40 days of training, both groups were performing only at approximately 75% - 85% correct.

Delayed spatial alternation

The percentages of correct trials (excluding correction trials) at each delay were averaged over blocks of 5 days and a three-factor repeated measures analysis of variance was again carried out on these data. The order of training was found to have had no significant effect on the overall performance on the delayed alternation task ($F(2,12)=0.11$, $p=0.90$), and none of the interactions involving the order of training factor was significant either (lesion treatment x order of training, $F(2,12)=0.92$, $p=0.43$; order of training x delays, $F(8,48)=1.02$, $p=0.44$; lesion treatment x order of training x delays, $F(8,48)=0.79$, $p=0.62$). For the subsequent analysis, therefore, the data from the three subgroups B1, B2, and B3 were pooled, and are summarised in Figure 52.

From the graph of percent correct responses against delay interval, it is clear that the two groups did not differ at all on any of the five delays, and that they showed a steady decrement in performance from the final block of 5 days on the nondelayed task, the means for which, pooled for comparison purposes, are presented as the first points on this graph, to the block of 10 secs delay sessions, when both groups were performing at chance level. A two-factor analysis of variance confirmed



*Final block of days on the nondelayed spatial alternation

Figure 52. Performance of normal and hippocampal pigeons on a delayed spatial alternation task.

these observations. The effects of the hippocampal lesions were not significant ($F(1,22)=0.0003$, $p=0.94$), and the effect of the delays was highly significant ($F(4,88)=38.95$, $p<0.00005$), but the lesion treatment x delays interaction was not significant ($F(4,88)=0.36$, $p=0.84$).

Discussion

The results of the first part of this experiment showed that pigeons with hippocampal lesions were not impaired in their performance on a spatial alternation task presented in an operant chamber. These results are therefore similar to those obtained by Stevens and Cowey (1972, 1973), and are in contrast to those reported by Niki (1966) and Riddell et al (1973). Although two-thirds of the pigeons in the present experiment had also been trained in either one or two other experiments, it is proposed that, because position was not the relevant cue in these other tasks, they were not incompatible with the learning of a spatial alternation. Indeed, it was found that prior training in the other experiments produced a practice effect for both groups, since their general level of performance improved with the prior training.

In the two-lever spatial alternation experiment reported by Stevens and Cowey (1972), the rats had previously been trained in a runway reinforcement shift experiment and in a reversal learning experiment. Although the nature of the reversal experiment is not specified, careful reading of a further paper (Stevens, 1973c) reveals that it was a serial position reversal task presented in a T-maze. However, no significant differences were found to occur between the hippocampal and the control rats, either on acquisition or on any of the twenty reversals. The first of these tasks is fairly obviously not likely to be incompatible with a spatial alternation problem, although this ordinarily would not be true of a spatial reversal task. However, in this case, not only were the rats given a large number of reversals, but the hippocampal rats performed as well as normal rats on each reversal. Furthermore, the task was

equivalent to a learning set experiment, since each reversal was presented daily, and only for ten trials, performance being measured in terms of the reduction in the number of errors over the twenty days. Thus, the animals were trained to respond equally to both sides over the twenty days, and also to shift responses from one side to the other relatively rapidly. In this case, therefore, it would appear that this type of prior training should not interfere with the learning of a spatial alternation. The rats that were trained on the spatial alternation task in an operant chamber by Stevens and Cowey (1973) had previously been tested in a spontaneous alternation experiment, and then on a 70:30 spatial probability discrimination in the same maze. The first of these clearly is compatible with a learned spatial alternation task, while the second task, although involving position as the relevant cue, nevertheless is one in which reinforcement is available from time to time in either goalbox in the T-maze, and it is therefore suggested again that prior training on such a task would be unlikely to produce large interference effects on the learning of a spatial alternation.

In contrast, the rats that were trained in a two-lever alternation task by Niki (1966) were first trained on the acquisition and then on the reversal of a position discrimination in a T-maze, and all the hippocampal rats were significantly impaired on the reversal. Also, the hippocampal rats that showed a large deficit in their performance on a two-lever alternation experiment that was reported by Riddell et al (1973) had previously been trained on a position discrimination in the same apparatus. These findings therefore suggest that hippocampal animals are only impaired on the acquisition of a spatial alternation in an operant chamber if they have been given prior training on a task which is likely to provide a high degree of interference.

That hippocampal animals appear to be more susceptible than normal animals to the effects of interference has been demonstrated in a variety of situations by a number of investigators. Thus, there is evidence that hippocampal rats are more likely to show

a passive avoidance deficit if previously given adequate approach pretraining (Stein and Kirkby, 1967), and that a DRL deficit does not occur unless the hippocampal animals are given CRF pretraining (Schmaltz and Isaacson, 1966b). Winocur and Mills (1970) have shown that preoperative training on a brightness discrimination had a greater interference effect on the postoperative learning of a pattern discrimination by hippocampal rats than it did on that of control rats, and more recently, Winocur (1979) found that, compared with normal rats, hippocampal rats show poorer acquisition or retention of a pattern discrimination following training on a high interference task.

The results of the second part of the present experiment showed that the performance of both hippocampal and normal pigeons deteriorated with increasing delays between trials, but that there were no differences between the two groups of pigeons. In those experiments in which a hippocampal deficit has been found to occur on a delayed spatial alternation problem, it is now fairly clear that the important feature of the task is the spatial factor rather than the delay (e.g., Mahut, 1971). In support of this notion is the finding that spatial alternation deficits in hippocampal animals are more likely to be found in an apparatus in which spatial cues are more prominent, and indeed, of the six studies of the effects of hippocampal lesions in rats on spatial alternation behaviour in a maze, in only one of these experiments did the hippocampal rats perform as well as the normal rats (Jarrard, 1975; see also O'Keefe and Nadel, 1978, p. 469). On the other hand, a spatial alternation deficit occurs less reliably in a WGTA (see the Introduction to this experiment), and appears to occur in an operant chamber only if the animals have been trained previously on an incompatible task. Thus, O'Keefe and Nadel (1978) have argued that, because of "the impoverished sensory environment of the Skinner box" (p. 318) only a limited amount of exploration occurs. Partly because of this, and also because food is always found in the same place, regardless of which lever is pressed, the usefulness of place hypotheses are minimised. Consequently,

the importance of the hippocampus, and therefore of the spatial information processing function that they ascribe to it, is minimised in an operant chamber. Hence, animals with hippocampal lesions should not be particularly affected compared with normal animals, since, it is argued, they both have to rely on the use of orientation hypotheses in order to learn the problem. This would perhaps appear to be discrepant with the finding, reported in Chapter 4, that hippocampal pigeons showed a serial position reversal deficit when trained in an operant chamber. However, it will be recalled that the main reason for their deficit was that they had difficulty in making sufficiently long runs of responses to the correct side in order to reach the learning criterion, and instead showed a greater tendency than the normal pigeons to shift their responses between the two keys. It would seem, therefore, that, while having a disruptive effect on the performance of a position habit, such a tendency would be beneficial to the acquisition of a spatial alternation. Consequently, the finding, in the present experiment, of normal performance by the hippocampal pigeons on a delayed spatial alternation task is not inconsistent with the serial position reversal deficit obtained earlier, and furthermore, the present result is similar to that found in hippocampal rats trained on a similar task.

CHAPTER 8 Acquisition of a Delayed Colour Alternation Task

Introduction

A number of investigators have studied the effects of hippocampal lesions in mammals on several types of alternation or sequential task. The various studies of spatial alternation have already been discussed in the previous chapter, in which it was reported that pigeons with hippocampal lesions were not impaired on either the nondelayed or the delayed task, and that similar effects have been found in hippocampal rats (Stevens and Cowey, 1972, 1973), cats (Brown et al, 1969), and monkeys (Waxler and Rosvold, 1970).

A second type of alternation problem is nonspatial alternation, but invariably this has been presented to hippocampal animals as a go, no-go task. The cued version of this task, in which a response is required to be made in the presence of one stimulus and to be withheld in the presence of the alternative stimulus, is usually referred to as a successive go, no-go discrimination. A review of these experiments has already been presented in Chapter 1, and it generally has been found that hippocampal animals are impaired on the acquisition of these tasks due to a reduced ability to withhold responses in the presence of the negative stimulus, although there have also been several reports in which the hippocampal animals performed as well as normal animals (Freeman et al, 1973; Gaffan, 1973; Freeman and Kramarcy, 1974; Plunkett and Faulds, 1979).

In the non-cued task, the animals are required to make a response on alternate trials and to withhold a response on the intervening trials. Of the various experiments that have been carried out using this procedure in an operant chamber, hippocampal deficits were reported in rats by Warburton (1969), Walker and Means (1973), and White (1974), while Means, Walker, and Isaacson (1970) and Walker, Means, and Isaacson (1970) reported superior performance in their hippocampal rats, and a similar facilitation was found in hippocampal cats by Brown et al (1969) and monkeys by

Mahut (1971) when trained in a WGTA. However, when the delay interval between trials was varied, Walker, Messer, Freund, and Means (1972) found that, with a 10 secs delay the hippocampal rats were superior, with a 20 secs delay they were no different from the normal rats, and with delays of 40 or 80 secs the hippocampal rats were impaired.

A further type of alternation task involves a response on one lever followed by a response on a second lever, and only the correct completion of the response sequence is rewarded. When Gross, Chorover, and Cohen (1965) trained hippocampal rats on this type of task, they found they were impaired. However, Jackson and Strong (1969) subsequently reported that hippocampal rats not only were not impaired on the acquisition of a two-lever alternating sequence, but were actually superior to normal rats on this and on the acquisition of higher order sequences of lever-press responses involving both two and three levers. A visual version of the task used by Gross et al, and in the first experiment of Jackson and Strong, had earlier been carried out by Kimble and Pribram (1963), with position as an irrelevant cue. Hippocampal monkeys were trained in an operant chamber containing sixteen press-panels arranged in a 4 x 4 array, and they were first required to respond in any order to two identical visual stimuli that were presented simultaneously on any two of the sixteen panels, in order to obtain a reward. Later, they were required to respond in a particular sequence to two different visual stimuli, which again were presented in random positions, for a reward. In both tasks the hippocampal monkeys were impaired, in the first task by repeating a response to the same panel, and in the second task by pressing the panels in the incorrect order. From these results, Kimble and Pribram concluded that "bilateral hippocampal lesions interfere selectively with the acquisition of behaviours which involve the execution of sequential responses" (p. 825).

However, as O'Keefe and Nadel (1978) point out, the true nonspatial analogue

of the spatial alternation task, which requires an animal to respond alternately to one stimulus on one trial, and to the opposite stimulus on the next trial, regardless of position, in order to obtain a reward on each trial, has not been presented to hippocampal animals. The most likely explanation for this is that it is an extremely difficult task, even for primates (see Williams, 1971a). In an experiment in which normal pigeons were trained with correction trials on a nonspatial (colour) alternation task such as this, Williams (1971a) found that very little learning, even after 50 days, occurred in those pigeons that were trained using either CRF or a FR5 schedule of reinforcement, their performance stabilising at about 65% correct. In contrast, pigeons trained on FR15 or FR30 schedules of reinforcement were performing at approximately 80% correct by day 20. Detailed analysis of the individual response data revealed that the pigeons trained on a FR5 schedule, or on CRF, tended to adopt strong position habits, and also that all pigeons showed a tendency to shift their responses between keys within trials, but this was much more marked for the pigeons on the higher FR schedules. Thus, Williams found that the predominant tendency was for the pigeons to repeat their response to the stimulus that was rewarded on the previous trial, but whereas the animals trained on the FR15 and FR30 schedules then corrected their response within the trial, those trained on CRF or a FR5 schedule did not. Subsequently, Williams (1971b) found that, by using the same procedure, and training pigeons on FR15 or FR30 schedules of reinforcement, they could acquire a delayed colour alternation (i.e., with position irrelevant) and were able to perform at above chance level even with a 45 secs delay.

The results of previous experiments reported in this thesis have suggested that, like the mammalian hippocampus, the avian hippocampus is not involved in the inhibition of response tendencies or of attention, or in the shifting of responses from one stimulus to another, and that hippocampal pigeons are capable of normal performance

on a delayed response task. Since the acquisition of a nonspatial alternation task requires the animal to attend to the relevant cue, to inhibit responses, and to shift its response to the other stimulus on each trial, the finding that hippocampal pigeons show normal performance on this task when trained with correction trials and a moderate FR schedule of reinforcement would provide further evidence to support these earlier findings. Furthermore, normal performance on a delayed nonspatial alternation task would also confirm the results obtained in the second part of the previous experiment, showing that pigeons with hippocampal lesions are not impaired on tasks involving delays.

Method

Subjects

Twelve pigeons with bilateral hippocampal lesions and twelve sham-operated or unoperated pigeons were used. They had all been trained in the two previous experiments which, together with the present experiment, were run in a balanced design. Full details of the design and of the pretraining that all pigeons underwent are presented in Chapter 6.

Apparatus

A standard Campden Instruments testing chamber was used, in which there was a white houselight and the keys could be lit with either red or green light.

Procedure

Nondelayed colour alternation training

Each training session began with the houselight on and both keys lit, one with red light and the other with green light, the positions of which were determined by Gellerman sequences. Five different sequences were prepared on punched tape (see Appendix), and they were used in conjunction with a small tape reader as before.

The response requirement was FR15 on either key, which then switched off both keylights. Responses on the two keys were counted by two separate predetermined counters and the outcome of a trial was determined by the key on which a total of fifteen responses were first made. FR15 on the correct key was reinforced with 3 secs access to food, while FR15 on the incorrect key was not food-reinforced, but instead turned off the houselight for 3 secs TO. Either event was followed by a brief (0.2 secs) delay interval, during which the predetermined counters were reset and, following a correct response, the tape-reader stepped the punched tape on to the next member of the Gellerman sequence, and then the keylights came on again for the next trial. Following an incorrect response a correction procedure was used in which the positions of the colours remained unchanged and the trial was repeated.

The correct colour and the key on which it was presented on the first trial of each day were varied according to a predetermined random sequence, and on subsequent trials the correct colour was the one which was not rewarded on the previous trial. Each pigeon was given 50 reinforced trials a day.

Delayed colour alternation training

On day 41 training on the delayed alternation task began, and the procedure was identical in all respects to that used in the nondelayed task, with the exception that the 0.2 secs interval was replaced by a variable delay interval. The values used, 1, 2, 3, 5, and 10 secs were the same as were used in the previous experiment, and the order of presentation was varied randomly on a day to day basis, as before. The pigeons were given 25 days of training so that they all received a total of five days on each of the five delay intervals.

Throughout the experiment electromechanical counters were used to record the numbers of correct and incorrect trials, together with the numbers of trials on which

15 responses were made on the red, green, left, and right keys. In addition, a Sodeco printout counter was used to record, first, the key on which the first response of each trial was made, and secondly, the key on which FR15 was completed, in order to allow an analysis of the response-shift behaviour of the two groups within trials to be carried out.

Results

Histology

Since these pigeons were also trained in the two previous experiments, the results of the histological analysis have already been presented in Chapter 6.

Nondelayed colour alternation

The data were averaged over blocks of 5 days, and again the effect of the order of training was examined using a three-factor repeated measures analysis of variance, which showed that the order in which the three subgroups of pigeons, C1, C2, and C3, were trained in the three experiments had no effect on their performance on the present task ($F(2,12)=0.44$, $p=0.66$). Since none of the interactions involving the order of training factor was significant either, the data from the three subgroups of eight pigeons each were combined so that subsequent analyses were carried out on two main groups of pigeons, one normal and one hippocampal, each consisting of twelve pigeons.

The mean percent correct scores (excluding correction trials) for the two groups are summarized in Figure 53. From this it can be seen that both groups were performing at below chance level for at least the first block of 5 days, but that they showed a steady improvement over the next seven blocks of days. However, there are two points which seem particularly noteworthy: the first is the close similarity in the performance of the two groups over the whole of the training period; the second is that, even after 40 days of training, both groups were performing at only 75% correct.

The analysis of variance that was carried out on these data confirmed the lack of a significant effect of lesion treatment on this task ($F(1,22)=0.26$, $p=0.62$) and that

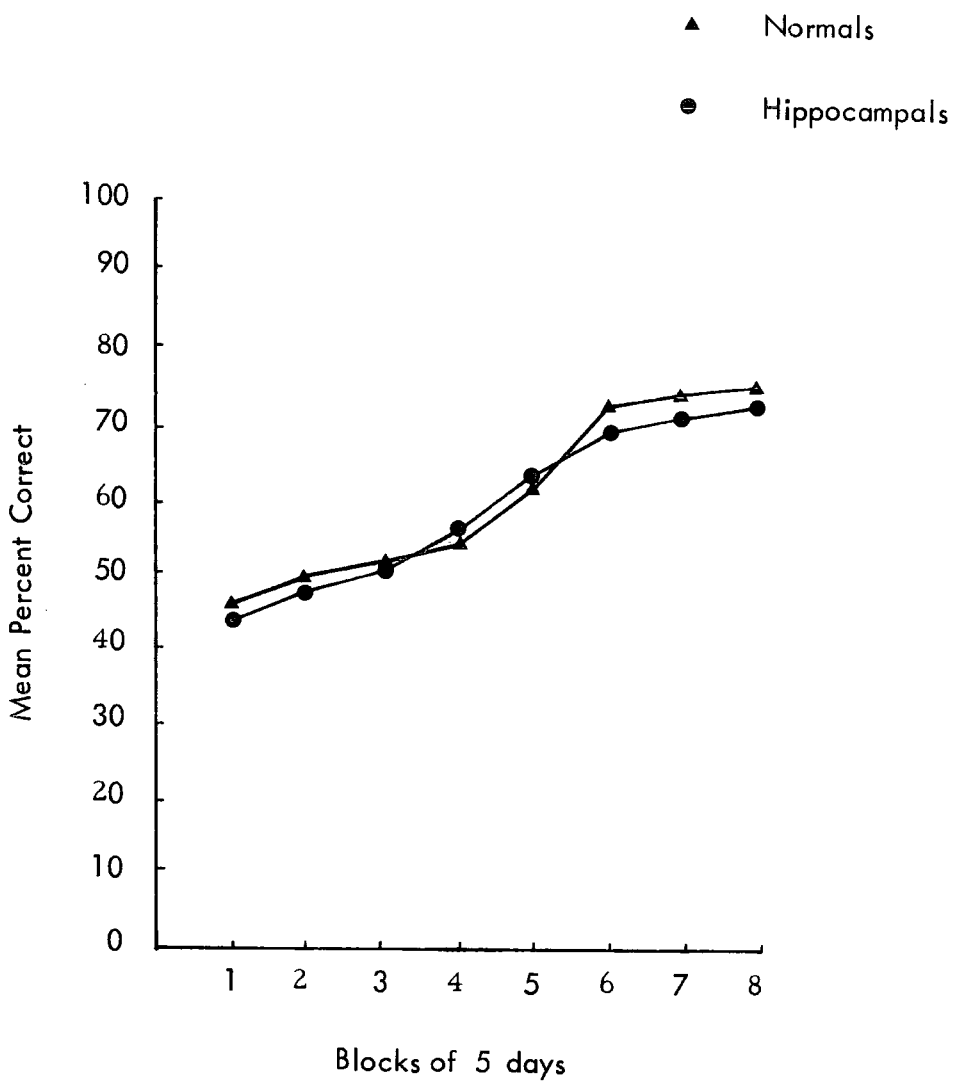


Figure 53. Performance of normal and hippocampal pigeons on a nondelayed colour alternation task.

the groups x blocks of days interaction was not significant ($F(7,154)=0.77$, $p=0.62$), but that the performance of the two groups showed a highly significant improvement over blocks of days ($F(7,154)=97.60$, $p<0.00005$).

Because of the vast amount of data that were obtained from the printout counter, the within-trials response-shift data were analysed for all pigeons over the first and the last five days only. However, two types of response-shift were distinguished:
a) a shift from a response to the incorrect colour (the colour responded to previously) at the beginning of the trial to the completion of FR15 on the correct colour, and
b) a shift from a response to the correct colour at the start of the trial to the completion of FR15 on the incorrect colour. These are summarised below as mean scores for each of the two blocks of 5 days for the two groups:

	Normals		Hippocampals	
Response Shift	First 5 Days	Last 5 Days	First 5 Days	Last 5 Days
Incorrect to correct	10.3	12.8	9.2	17.6
Correct to incorrect	1.6	4.3	1.3	3.7

A three-factor analysis of variance with repeated measures was carried out on the scores for the individual days, and it was found that there was no significant difference overall between the two groups ($F(1,22)=0.27$, $p=0.61$), but the difference between the scores on the first 5 days and on the last 5 days was highly significant ($F(9,198)=10.59$, $p<0.00005$), and the difference between the scores on the two types of response-shift was also highly significant ($F(1,22)=44.65$, $p<0.00005$). However, none of the interactions was significant (all F's <1.62, all p's >0.11). Thus, together the two groups of pigeons showed a significant increase during training in their tendency to correct their errors within a trial, and although there was not a significant difference between the two groups, the mean scores for the final block of 5 days suggest that the

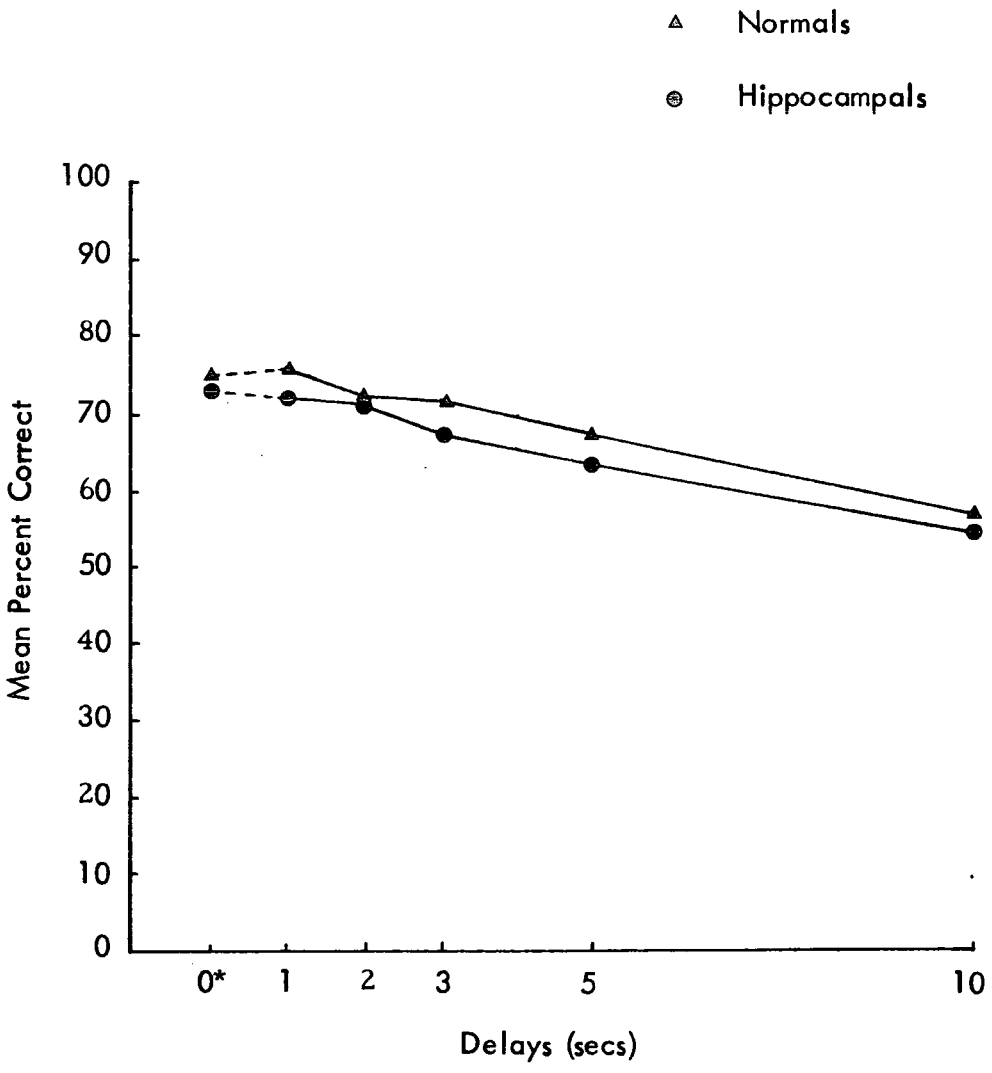
hippocampal pigeons had a greater tendency to shift responses, and thereby correct their errors. On the other hand, it is clear that relatively few shifts from a potentially correct to an incorrect response were made by either group, that they both showed a small, but insignificant increase in their tendency to make this type of response-shift within a trial, and that the two groups did not differ at all on this measure.

Delayed colour alternation

The mean percent correct trials were analysed over blocks of 5 daily sessions for each of the five delay periods, with the order of training again being considered as a separate factor, and as before, no significant differences were found between the three subgroups ($F(2,12)=0.23$, $p=0.80$), and none of the interactions involving the order of training factor was significant (all $F's<1.5$, all $p's>0.19$). The data from the three subgroups were pooled in the subsequent analysis and are summarised in Figure 54. The two groups of pigeons performed as accurately at the 1 sec delay as they had done in the final block of days on the nondelayed task, but in the subsequent delays their level of accuracy declined progressively until, at a delay of 10 secs, their performance was not much above chance level (mean scores: normal group, 57.4% correct; hippocampal group, 55.4% correct). Furthermore, over the whole range of the delays used, the hippocampal group gained consistently lower scores than the normal group. An analysis of variance, however, showed that the main effect of hippocampal lesions was not significant ($F(1,22)=3.15$, $p=0.08$), and that the interaction of lesion treatment x delays was also not significant ($F(4,88)=0.20$, $p=0.94$), but that the reduction in accuracy with increasing delays was highly significant ($F(4,88)=30.91$, $p<0.00005$).

Discussion

The results of this experiment show quite clearly that the hippocampal pigeons were not impaired on the acquisition of a nonspatial alternation task presented in an



*Final block of days on the nondelayed colour alternation.

Figure 54. Performance of normal and hippocampal pigeons on a delayed colour alternation task.

operant chamber, indicating that they are capable of normal levels of attention to the relevant cue. This was confirmed by an analysis of the responses made by each pigeon on each trial during the first and the last blocks of 5 days, which also showed that the hippocampal pigeons were as able as the normal pigeons to inhibit inappropriate responses and to shift their responses to the appropriate key. The finding that, during training, both groups of pigeons increased their tendency to shift responses from the incorrect to the correct key was also obtained by Williams (1971a), who reported that this type of within-trial response-shift was correlated with learning efficiency, increasing most during the period of most rapid learning. These results therefore support those obtained in the earlier experiments that are reported here, confirming that the avian hippocampal deficit, like the deficit found following hippocampal lesions in mammals, cannot be readily explained in terms of increased response-perseveration, impaired attentional processes, or an impaired ability to shift responses (see especially Chapters 3, 4, and 6).

The performance of the two groups of pigeons on the delayed alternation task was again very similar, confirming the findings from the previous experiment that hippocampal pigeons are not impaired on tasks involving delays between trials, a result that has been obtained in hippocampal mammals by others (see Chapter 7). The present results, then, together with those that were reported in the previous chapter, have shown that hippocampal lesions in pigeons do not impair their ability to acquire either a spatial or a nonspatial alternation task, with or without delays, at least up to 10 secs, when presented in an operant chamber, and since comparable results have also been reported to occur in hippocampal mammals (Brown et al, 1969; Waxler and Rosvold, 1970; Stevens and Cowey, 1972, 1973) it would appear that, once again, the present results suggest that the hippocampus in birds and mammals have similar functions.

CHAPTER 9 DRL 10 Performance and the Distribution of Interresponse Times

Introduction

In the DRL 10 experiment (Chapter 5) the intention had been to record interresponse times (IRTs), but owing to equipment failure this proved to be not possible. However, there arose subsequently the opportunity to use a data-logging device, which allowed for more reliable and accurate recording of IRTs, although unfortunately the equipment was available for only the relatively short period of approximately ten days. Nevertheless, on the basis of the data obtained earlier, it seemed reasonable to assume that a temporal discrimination would have developed by the end of this period. Furthermore, Ellen et al (1973) trained hippocampal lesioned rats on a DRL 20 schedule for 15 days, following CRF training, and from their results it is clear that by the tenth day the numbers of responses made by the several groups had become fairly stable, and for most of the groups the numbers of reinforcements obtained had also begun to stabilise, although this would appear to be less important, since Ellen et al found that the IRT data resembled the response data more closely than they did the reinforcement data.

There are two reasons why an analysis of IRTs is of interest. First, Kramer and Rilling (1970), in a comprehensive review of DRL studies, recommended that, in addition to the measures of number of responses, number of reinforcements, and percent reinforced responses (efficiency ratio), studies of DRL performance should include at least one IRT analysis. Preferably the interresponse time per opportunity (IRT/OP) measure proposed by Anger (1956) should be used, but ideally both IRT analyses should be presented, the measure more commonly used being the relative frequency of IRTs (the number of responses in a particular IRT class expressed as a percentage of the total responses in all IRT classes). One of the main advantages of the IRT/OP analysis is that it is much more likely to detect the presence of a temporal discrimination than is the relative frequency measure, but, as Kramer and Rilling point out, it has other advantages

too, one of them being that, if responses are random with respect to time, the probability of all IRT/OP values should be equal. The need for the presentation of both response measures and IRT measures is to allow more reliable comparisons to be made between studies, since, even on the same DRL schedule, both response rate and reinforcement rate can show considerable variation. Although, as was pointed out in Chapter 5, the efficiency ratio enables comparisons to be made across studies, it does not provide adequate information about the nature of the temporal discrimination. Increases in efficiency ratio over days have been assumed to reflect an increase in the number of IRTs that exceed the critical value for the particular DRL schedule, but, as noted above, Ellen et al (1973) found that changes in the slopes of the IRT/OP distributions with training were not consistently related to the number of reinforcements obtained. Thus, Kramer and Rilling argue that "for the sake of clarity, completeness, and ease of comparison with other experiments, all DRL studies should include at least one IRT analysis, if not both" (p. 230).

The second reason for recording IRTs is that temporal discrimination in hippocampal animals trained on a DRL schedule, a task that has been employed in more than twenty studies, could therefore be analysed. But, as Haddad and Rabe (1969) noted, "temporal analyses of operant responding have been uncommon for rats with hippocampal lesions" (p. 311). Among those few who have presented IRT measures in DRL studies with hippocampal animals are Ellen et al (1964, 1970, 1973), MacDougall, Van Hoesen, and Mitchell (1969), and Johnson et al (1977). Nevertheless, Clark and Isaacson (1965), in one of the earliest studies of DRL performance, suggested that, because hippocampal rats show impaired learning of a passive avoidance task, and greater resistance to extinction, they ought to have difficulty in acquiring the temporal discrimination required by a DRL schedule. Shortly afterwards, Schmaltz and Isaacson (1966) also suggested that hippocampal rats might be impaired in their ability to acquire

a temporal discrimination, but they found that those animals that had not received CRF training prior to DRL training were not deficient compared with normal rats, and therefore they assumed that hippocampal rats do not suffer from an impaired ability to form a temporal discrimination. However, in neither of these experiments were IRT analyses presented.

That mammals with hippocampal lesions show some alterations in timing behaviour compared with normal animals was first demonstrated, in fact, by Ellen and Powell in 1962. They trained hippocampal and normal rats on a FI60 schedule of reinforcement and found that, although the hippocampal rats showed fairly normal FI performance as a whole, they actually had a lower response rate than the normal rats, took longer to develop the typical post-reinforcement pause (PRP), and made fewer responses immediately prior to a reinforcement. A similar effect was also reported by Beatty and Schwartzbaum (1968). On the other hand, Haddad and Rabe (1969) trained hippocampal rats on a FI60 schedule but found that they had a significantly higher response rate just prior to reinforcement without showing any deficit in their ability to develop a PRP, and this effect was also found to occur in squirrel monkeys with hippocampal lesions trained on a FI5 mins schedule (Jackson and Gergen, 1970). Although it is not easy to reconcile these two sets of results, it is possible that procedural differences contributed partly to these effects, and there is also the suggestion that differences in the locus of the lesion are partly responsible. While the rats in the experiment by Haddad and Rabe (1969) had large lesions involving both the anterior and the posterior hippocampus, and the monkeys of Jackson and Gergen (1970) had lesions in the posteroventral region of the hippocampus, Ellen and Powell (1962) gave their rats anterior hippocampal lesions. Similarly, the rats in the experiment by Beatty and Schwartzbaum (1968) had received large anterior hippocampal lesions although they included some posterior hippocampal damage. The suggestion that there may be

functional differences between the two regions of the hippocampus have already been made and supported by, for example, Nadel (1968), Stevens and Cowey (1973), and Johnson et al (1977).

An analysis of IRTs in a DRL experiment on rats with hippocampal lesions was first reported by Ellen et al (1964), although they found there to be no obvious effects of the lesions. MacDougall et al (1969) trained rats preoperatively on a DRL 20 schedule and then compared this with their subsequent postoperative performance. Rats with large lesions of the fimbria and fornix were found to show a marked increase in short-latency responses (0-4 secs) as well as in longer latency responses (4-12 secs) during the first six postoperative days. Over days 7-12 they showed a small reduction in the relative frequency of shorter IRTs, but in both blocks of days they had a significantly higher response rate than the operated control animals, and made a significantly lower percentage of reinforced responses. Furthermore, their IRT/OP distribution was much flatter than that of the control rats, and they did not show the typical bimodal distribution, with the first mode in the shortest category of IRTs. This therefore demonstrated much more clearly than the relative frequency analysis that these animals were impaired in their ability to develop a temporal discrimination, and also that they were less likely than the normal rats to make short latency responses immediately following a reinforcement.

Ellen and Aitken (1970) trained groups of rats with either anterior or posterior hippocampal lesions on a DRL 20 schedule and found that neither group was impaired, either on the response measures, or on the relative frequency of IRTs measure, although they did not present an IRT/OP analysis. Later, Ellen et al (1973) trained rats with either anterior or combined anterior and posterior hippocampal lesions on a DRL 20 schedule, and found that only the rats with the combined lesions that had received prolonged CRF training had a markedly higher response rate. These animals were also

the only ones in which the IRT/OP analysis, in which 1 sec categories were now used, showed a temporal discrimination deficit, even after fifteen days of training. However, both hippocampal groups obtained significantly fewer reinforcements than the control group.

Finally, Johnson et al (1977) also trained rats preoperatively on a DRL 20 schedule, and then compared their pre- and postoperative performances. They, too, found that, during postoperative days 1-5, total fornix lesions produced a significant reduction in the percent of reinforced responses made, and also an increase in IRTs/OP in the 4-8, 8-12, and 12-16 secs categories, in the same way that anterodorsal hippocampal lesions did in their experiment. But, unlike the anterodorsal hippocampal group, who regained some of their temporal discrimination ability by days 26-30, the total fornix group showed no evidence of successful temporal discrimination, thereby confirming the finding of MacDougall et al (1969), and demonstrating a functional difference between the effects of total fornix lesions and lesions of the anterodorsal hippocampus. Nevertheless, the anterodorsal hippocampal rats were still impaired. In contrast, they also found that rats with posterior hippocampal lesions did not show a deficit, either in their efficiency scores, or in the IRT/OP analysis.

Although somewhat confusing and contradictory, there is obviously some evidence which shows that, under certain conditions, mammals with hippocampal lesions are impaired in their ability to develop an appropriate temporal discrimination, in both FI and DRL schedules, and also that there is not necessarily a good correlation between the IRT/OP distribution and the number of reinforcements gained. In the previous DRL 10 experiment, a deficit was found in the hippocampal pigeons similar to that which has been described in hippocampal rats and monkeys. The present experiment attempted to replicate that finding, and also to provide some detailed data on the temporal discriminatory performance of normal and hippocampal pigeons

using both the relative frequency of IRTs and the IRT/OP analyses.

Method

Subjects

Twelve pigeons were used, six sham-operated or unoperated controls and six with bilateral hippocampal lesions. All twelve animals had been tested in three previous experiments (visual form discrimination, delayed spatial alternation, and delayed colour alternation tasks). Following completion of these three experiments all twenty-four pigeons were returned to ad lib feeding for approximately four months. Six pigeons from each of the two groups (Normals: Numbers 76, 80, 81, 82, 84, 92 and 96; Hippocampals: Numbers 73, 75, 83, 85, 86 and 98) were then selected at random from the twenty-four, and were again selectively deprived of food in order to reduce them to 80% of their free-feeding weights in preparation for the present experiment.

Apparatus

A standard two-key Campden Instruments operant chamber was used, in which the right-hand key was blanked off as before, and the left-hand key could be illuminated with white light.

Procedure

Because of the extensive training that these pigeons had already received, it was felt unnecessary to give them explicit CRF pretraining prior to DRL training. However, they were each given minimal retraining to peck the key and to obtain food for 3 secs. This was followed the next day by training on a DRL 10 schedule, the procedure for which was identical to that described in Chapter 5. Each daily session was again of 20 mins duration, the end of which was signalled by the keylight being switched off, and subsequent responses had no effect. The total numbers of responses made and reinforcements received during DRL training, or of IRTs $\geq$10 secs achieved in extinction, were recorded on electromechanical counters. In addition, IRTs were recorded in 0.2 secs intervals using a data-logging device which received an input from the response

key via the standard Campden Instruments control panel and a pulse-former in the modular programming equipment that was used to control the experiment. The output of the device was recorded on magnetic audio-tapes running at $1\,^{7}/_{8}$ ips on a standard Sony two-track stereo tape recorder, and the data were transferred on to computer files for subsequent analysis by playing the recorded tapes back at 15 ips through an analog-to-digital converter (ADC). As noted in the Introduction to this experiment, the data-logger was available only for a limited period. The pigeons were therefore given ten days of DRL training, followed on the eleventh day by a single 20 mins extinction session, as before.

Results

Histology

The reconstructions of the lesions in these hippocampal pigeons have already been presented in Chapter 6.

DRL training

1 Responses, reinforcements, and efficiency

From the outset both groups of pigeons showed a marked tendency to overrespond, but this was particularly exaggerated in the hippocampal pigeons. The mean daily responses made by each group are presented in Figure 55, from which it can be seen that the number of responses made by the normal pigeons was approximately constant over the ten days (mean, 289.3). On the other hand the hippocampal pigeons made considerably more responses than the normal pigeons on each of the first three days (mean, 748.5), subsequently reducing their response rate to a level that was more similar to that of the normal group, and roughly maintaining it until the end of DRL training (mean over days 4-10, 454.8). Analysis of variance revealed that the overall difference between the two groups was highly significant ($F(1,10)=24.69$, $p<0.001$), that there was a significant reduction in responses over the ten days ($F(9,90)=4.53$,

$p = 0.0001$), and that the interaction of the groups x days was also highly significant ($F(9,90) = 5.42$, $p < 0.00005$).

The second measure, summarised in Figure 56, is the mean reinforcements obtained daily by each group, and it can be seen that, although the scores for the hippocampal group are consistently below those for the normal group, the differences between the two groups do not appear to be particularly large. However, the score for the hippocampal group decreases over days, whereas the number of reinforcements gained by the normal pigeons shows a tendency to increase slightly over days. The analysis of variance that was carried out on these data showed that the difference between the groups was not significant ($F(1,10) = 3.28$, $p = 0.097$), the effect over days was not significant ($F(9,90) = 1.27$, $p = 0.26$), and that the groups x days interaction was also not signicant ($F(9,90) = 1.85$, $p = 0.069$).

The efficiency ratio, or percent reinforced responses, was calculated as before and is summarised in Figure 57. From this it can be seen that the hippocampal pigeons made no more than about 6% reinforced responses, although their performance was generally somewhat below this (mean, 3.7%). In comparison, the normal pigeons obtained a maximum efficiency score of 18.7%, although their mean efficiency was 9.8%. Also, while the normal pigeons showed a slight tendency to improve their efficiency over the ten days, the hippocampal pigeons maintained a fairly constant level of performance and showed no improvement at all. An analysis of variance confirmed that the normal group made a significantly greater proportion of reinforced responses than the hippocampal group ($F(1,10) = 6.87$, $p < 0.025$), but showed that there was not a significant effect over days ($F(9,90) = 0.80$, $p = 0.62$), and that the interaction between the groups over days was not significant ($F(9,90) = 1.41$, $p = 0.19$).

II IRT analysis

When the total numbers of IRTs and of IRTs $\geq$ 10 secs per pigeon per day were

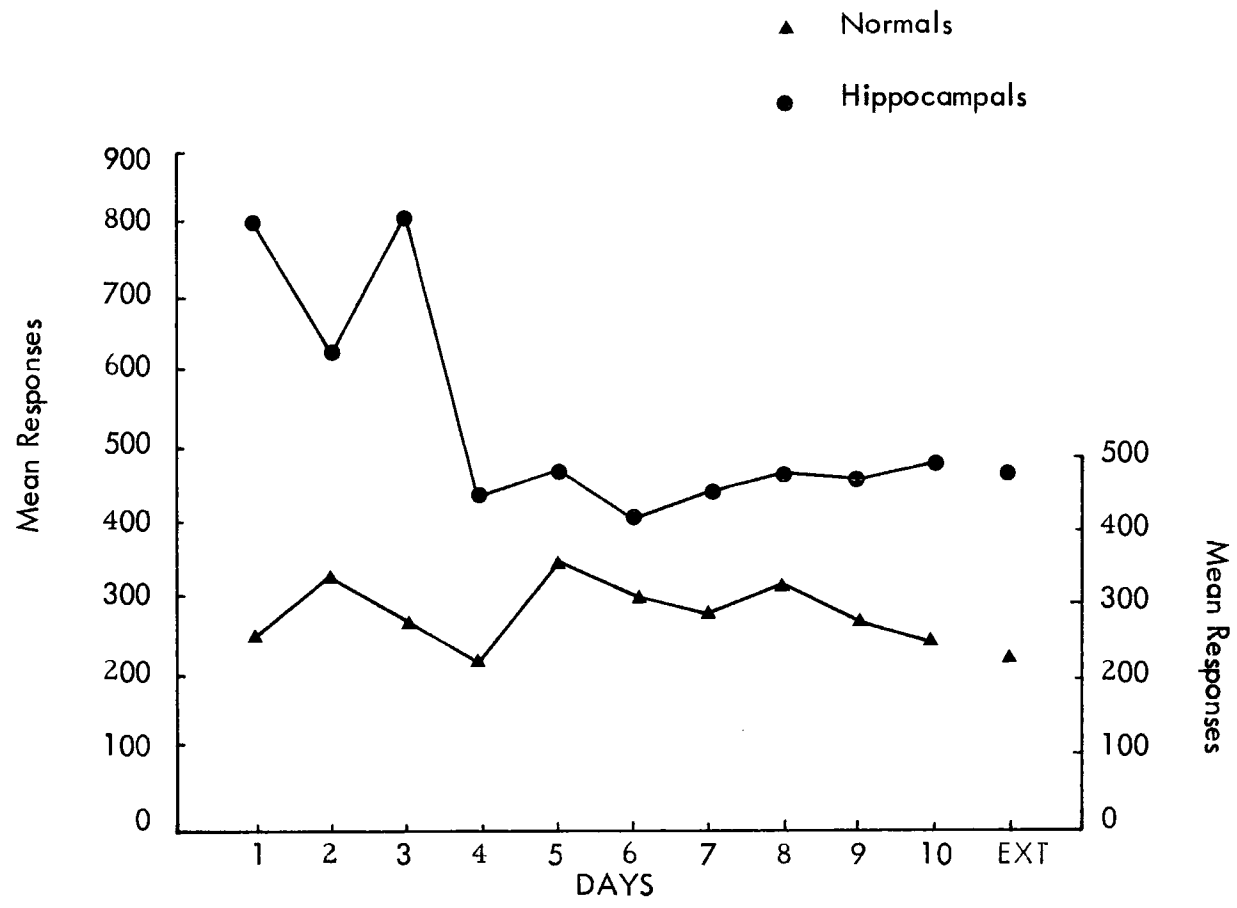


Figure 55. The mean numbers of responses made over the 10 days of DRL 10 training, and in the single extinction session (EXT).

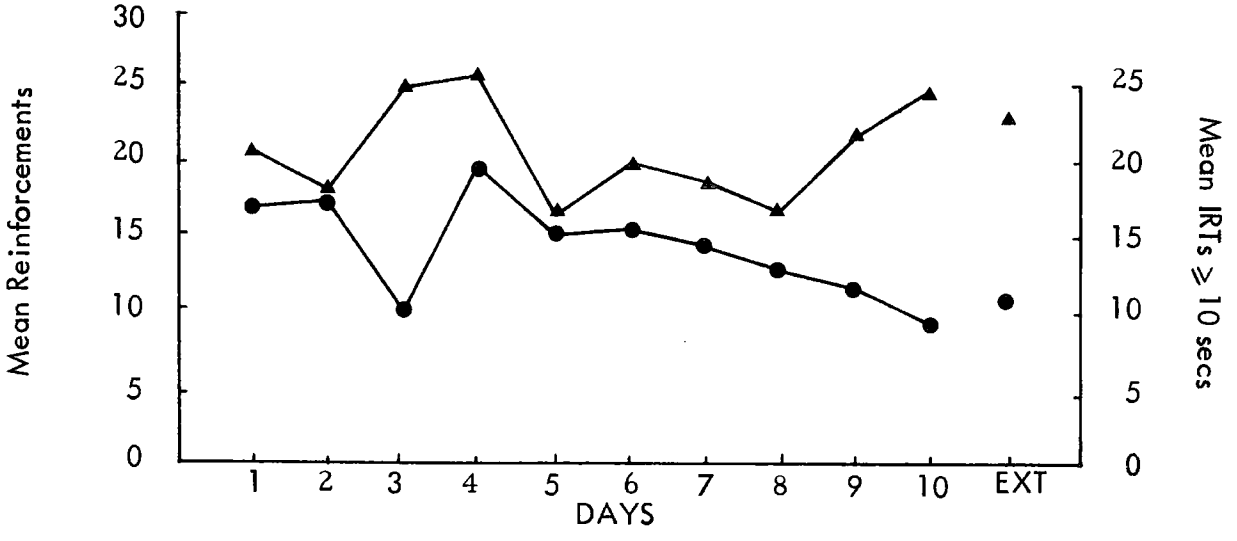


Figure 56. The mean numbers of reinforcements obtained over the 10 days of DRL 10 training, and the number of IRTs $\geq$10 secs in the single extinction session (EXT).

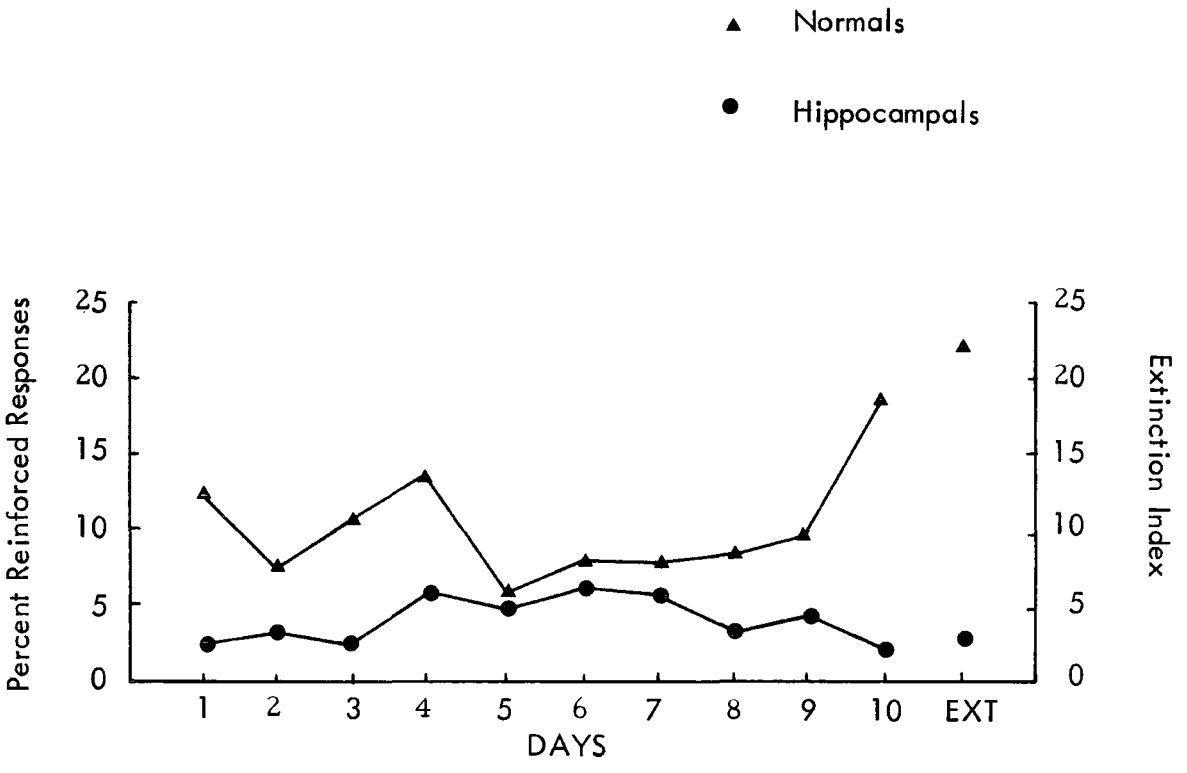


Figure 57. The mean percentage of reinforced responses over the 10 days of DRL 10 training, and the mean extinction index in the single extinction session (EXT).

obtained from the computer analysis of the IRT data and compared with the corresponding numbers of responses and reinforcements recorded by the electromechanical counters, it was found that, effectively, the data-logger consistently overestimated the numbers of IRTs $\geqslant$ 10 secs, and therefore reinforced responses, by approximately 15%. This was undoubtedly due mainly to the small degree of inaccuracy inherent in the particular timers used to control the DRL interval and the reinforcement duration, and in fact was most likely due to error in the calibration of the controls on the timers. However, since the degree of error remained fairly constant across both pigeons and days, it can be safely disregarded. Nevertheless, it should be noted that the responses, reinforcements, and efficiency ratio analyses relied on the data recorded by the electromechanical counters, while the IRT analyses used the data recorded by the data-logger.

a) Relative frequency of IRTs

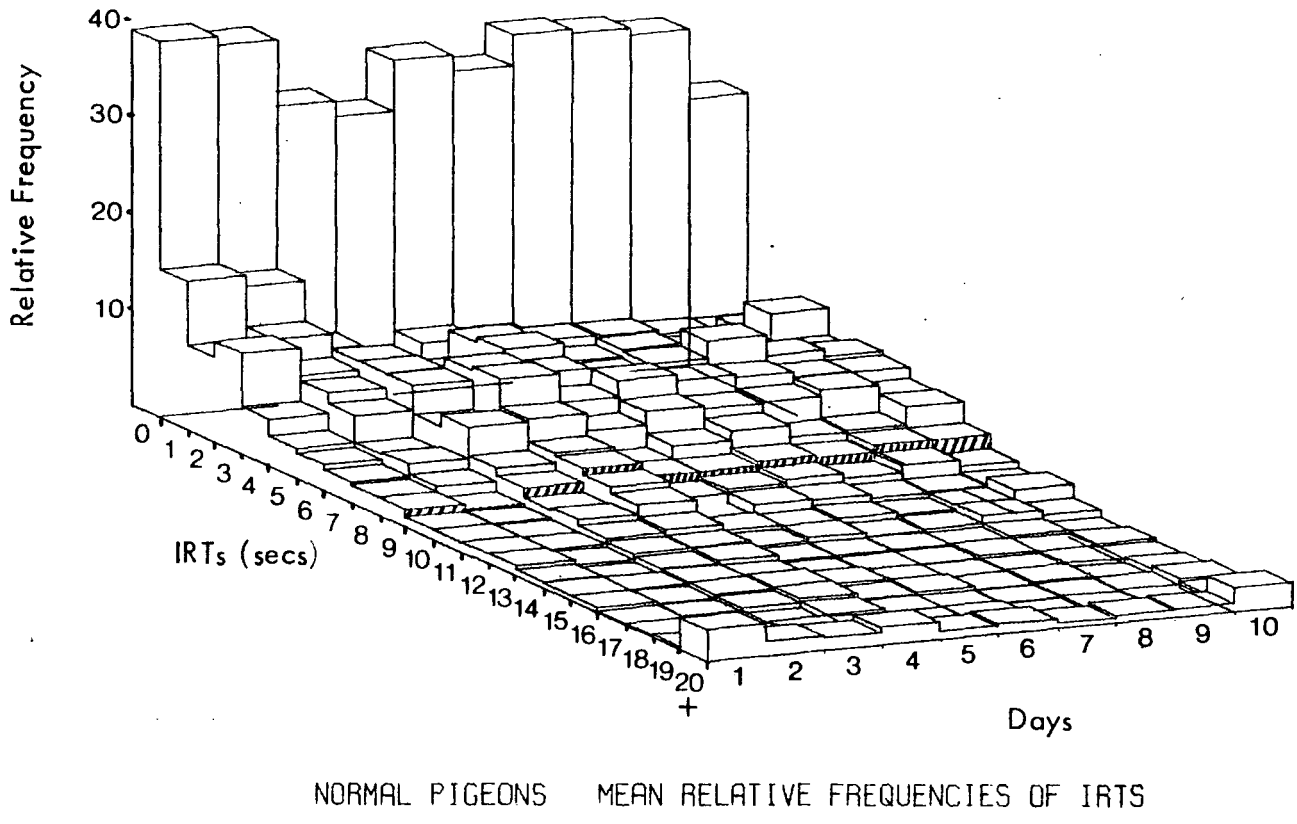
The IRTs, recorded in 0.2 secs intervals, were grouped into 1 sec class intervals for the relative frequency analysis, but since few responses tended to be longer than 20 secs, all of these longer IRTs were binned into a single category. Thus, there were twenty 1 sec IRT classes, from 0-19.8 secs (0-0.8, 1.0-1.8, 1.0-2.8 secs, etc.), and a final class consisting of all IRTs that were 20 secs and longer.

Changes in IRT distributions typically occur over a period of DRL training and it is not uncommon for animals to be given extended training to enable a stable performance to be established before IRT distributions are obtained. However, since this was not possible in the present experiment, it seemed appropriate instead to present the relative frequencies of responses in the various IRT classes for each of the ten days of training. In this way it was hoped that any changes in IRT distribution that occurred, reflecting the development of a temporal discrimination, might become more apparent. For this purpose the SYMVU Harvard computer graphics program was used in conjunction with the NUMAC graphical subroutine library and the data were plotted in the form of a three-dimensional histogram for each of the two groups of pigeons. These are presented

in Figure 58, and from this a number of tendencies, and a number of differences between the two groups may be observed.

Both groups typically made large numbers of short (0-0.8secs) IRT responses, which Sidman (1956) has called 'bursts'. The relative frequency of these burst responses for the normal group remained roughly constant over the ten days of training, whereas for the hippocampal group they were reduced abruptly on day 4, with a further smaller reduction after day 5, although throughout the ten days the hippocampal pigeons made more burst responses compared with the normal pigeons; the differences, however, were rather small on days 6 and 7 (hippocampal pigeons, 35.8% on day 6 and 38.6% on day 7; normal pigeons, 33.5% on day 6 and 36.8% on day 7). The hippocampal pigeons also made noticeably more responses in the 1.0-1.8 secs category on each day compared with the normal pigeons, and on the whole, the relative frequencies in this category remained fairly constant for the hippocampal pigeons, although there were clearly some fluctuations from day to day. However, for the normal group the relative frequency of responses in this category showed a small but consistent decline over days. In contrast, it appears that, in both groups, the longer (4.0-10.0 secs) IRTs showed a gradual increase in relative frequency from day 1 to day 10, although the increase in 8.0-10.0 secs IRTs was not as marked in the hippocampal group as it was in the normal group.

Once stable responding on a DRL task is reached, the relative frequency curve more often than not is bimodal, with the first peak occurring at the shortest IRTs, i.e., burst responses. This is followed by a minimum which may extend over several IRT categories, after which the curve rises to a second maximum which is usually at the shortest IRT that is reinforced. From Figure 58 it can be seen that a bimodal relative frequency curve began to appear in the normal data on day 4, and was maintained up to and including day 10. As noted above, however, since ten days of training was insufficient



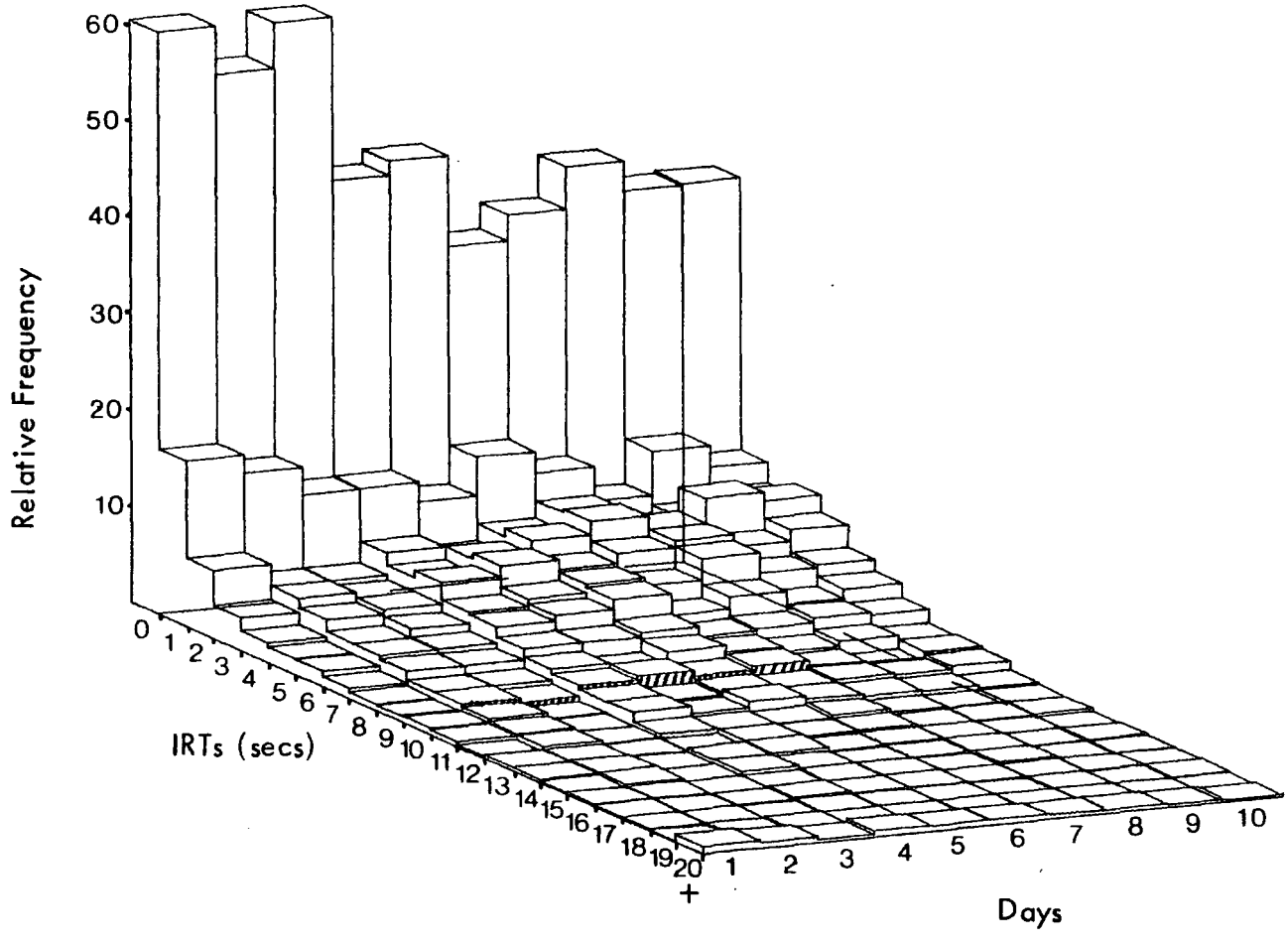


Figure 58. Distributions of relative frequencies of IRTs on each of the 10 days of DRL 10 training.

to allow stable responding to occur, the second peak on any of the days was at 3.0-3.8 secs, considerably shorter IRTs than the minimum response interval that was reinforced. In contrast, a similar bimodal distribution does not appear to have developed in the hippocampal data, even by the tenth day of training.

Finally, it should be noted that, although the differences were small, the normal animals tended to achieve more IRTs longer than 10 secs than the hippocampal animals on all ten days, and this was particularly noticeable in the 20 secs category. In order to show more clearly the changes that occurred over the ten days for both groups, and the differences between the groups at the beginning and end of training, separate relative frequency histograms for the two groups on day 1 and day 10 are presented in Figure 59.

From the relative frequency analysis it appears, first, that the hippocampal group made considerably more shorter and fewer longer IRT responses than the normal group over the whole of the DRL training period, and secondly, that while a temporal discrimination was beginning to emerge by the tenth day for the normal pigeons (see Figure 59), their responses were not yet under the control of the 10 secs DRL interval. On the other hand, no such rudimentary temporal discrimination had begun to appear by the tenth day in the responses of the hippocampal group.

b) IRTs/OP

Anger (1956) and Kramer and Rilling (1970) have suggested that the IRT/OP analysis is both more appropriate and more sensitive than the relative frequency analysis, since the number of different IRTs that are available in any particular trial depend on the animal's behaviour in that trial. For an IRT to occur in a given category the animal must withhold a response for at least as long as the minimum value of that IRT category, and once the response has occurred it necessarily precludes the occurrence of a longer IRT on that trial. Thus, it may be said that the time during which a response is withheld

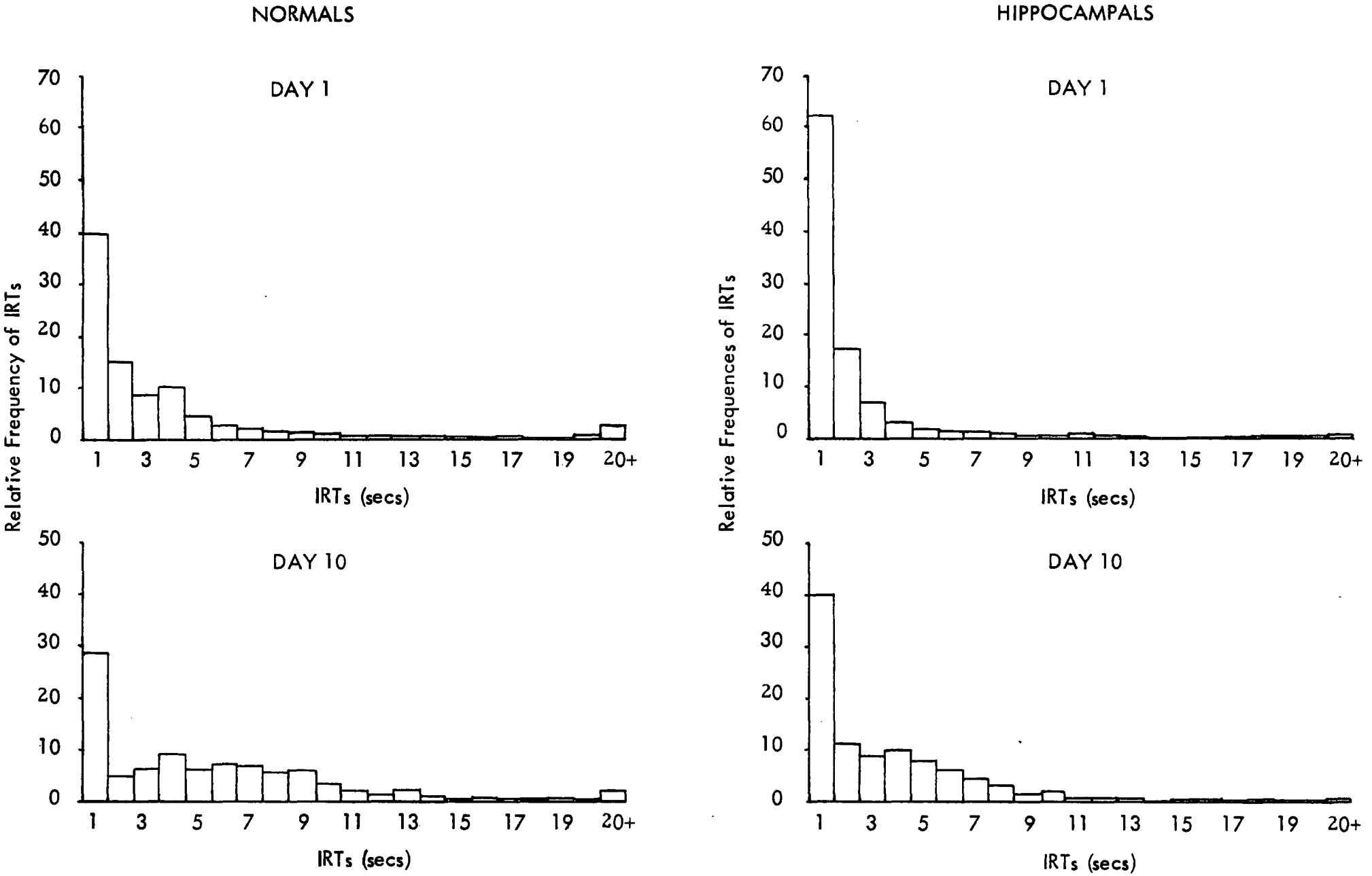


Figure 59. Distributions of the Relative Frequencies of IRTs on Day 1 and Day 10.

provides an opportunity for the IRT . The number of opportunities for a response in a particular IRT category is equal to the number of responses in that category, plus the number of responses with longer IRTs, and therefore there are more opportunities for short IRTs than for long IRTs. It is for this reason that the relative frequency analysis is likely to be misleading, but the different IRT categories can be equated by calculating the number of responses in a category as a proportion of the opportunities for that category - the interresponse times per opportunity, or IRTs/OP measure (Anger, 1956), and this provides an estimate of the probability of a response occurring in a givenIRT category.

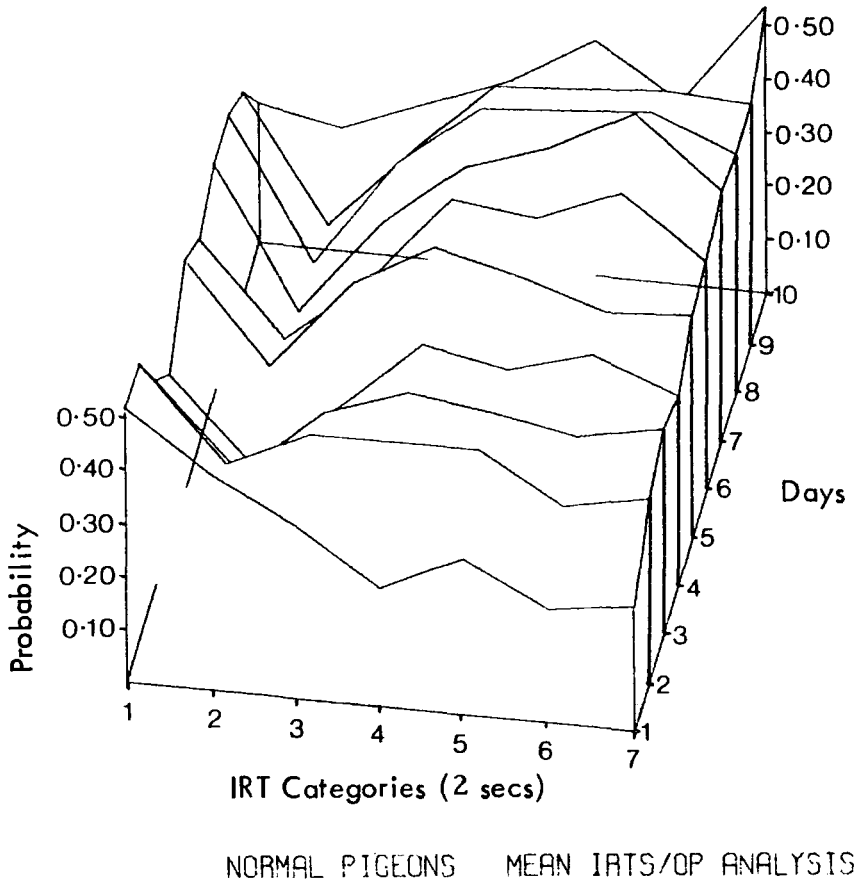
This measure has a number of advantages, which have already been referred to in the Introduction to this experiment, but there is a difficulty, too, which does not occur in the relative frequency analysis. This refers to the fact that, with increasing IRT length there is necessarily a decrease in the number of opportunities, i.e., the sample size, from which to derive the IRT/OP values. Therefore, with increasing IRTs the reliability of the measure decreases, and it is important to restrict the IRT/OP analysis to those IRT categories in which a certain minimum number of opportunities per session occur. For the IRT/OP analysis in this experiment the IRT data were grouped into 2 secs categories (0.0-1.8, 2.0-3.8 secs, etc), and to avoid the problem of unreliable estimates of IRTs/OP due to inadequate sample size, values were not obtained for IRTs longer than 14 secs since, in most cases, fewer than 15 opportunities occurred in longer IRT categories.

Using the SYMVU computer graphics program, daily IRT/OP distributions were plotted on a three-dimensional graph for each group, and they are presented in Figure 60. It is now quite apparent that a typical bimodal curve occurred in both groups, becoming established on the second day for the normal group, and on the third day for the hippocampal group. In addition, it appears that, on day 4 for both the

normal group and the hippocampal group, the second peak began to occur at the 10.00-11.8 secs category. The IRT/OP analysis plainly demonstrates, therefore, that both groups had begun to form a temporal discrimination within the rather limited training period that was available to them, and that the responses of both groups of pigeons were under the control of the 10 secs DRL interval.

Further observations that can be made from Figure 60 are, first, that the hippocampal pigeons, on the whole, showed a greater probability of making a response in each of the IRT categories on each of the ten days, compared with the normal pigeons; secondly, that after day 5 the slopes of the probability curves between the first mode, in the shortest IRT category, and the minimum, which invariably occurred in the next IRT category, were generally greater for the normal pigeons (although an obvious exception to this occurs on the final day of training); and thirdly, that the curves for the hippocampal pigeons tended to be flatter than those for the normal pigeons. These comparisons between the two groups indicate that, when the opportunities to respond were taken into account, the hippocampal group tended to make more responses in most categories, including longer IRTs, and that the responding of the hippocampal group was under less control of the 10 secs DRL interval, and therefore the reinforcement contingencies, than was that of the normal group.

The IRT/OP distributions are presented for each of the ten days for each group because the training period that was available to the pigeons was not long enough to allow the patterns of responding to become stabilised, and it was also for this reason that the data have been presented as mean IRTs/OP for each of the two groups. One possible disadvantage of this procedure, however, is that an averaged IRT/OP distribution may become distorted by the contribution of a few atypical values in particular IRT categories. For this reason, therefore, it was felt necessary to include IRT/OP distributions on each of the ten days for two typical pigeons, one from each group.



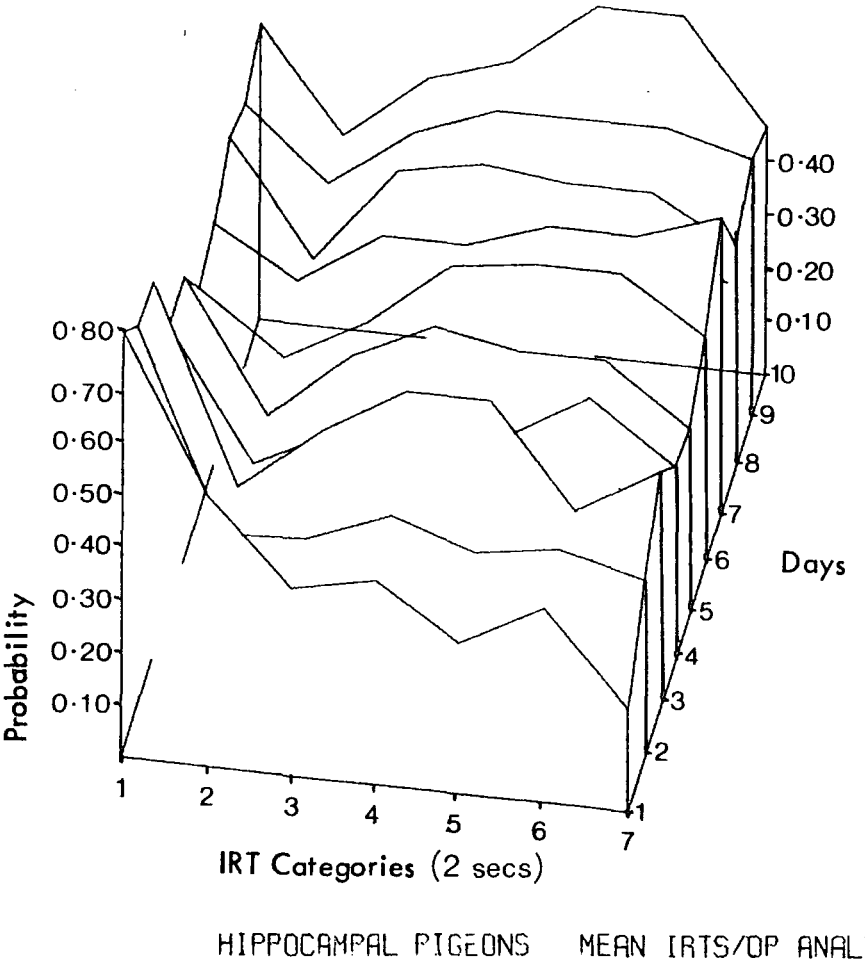
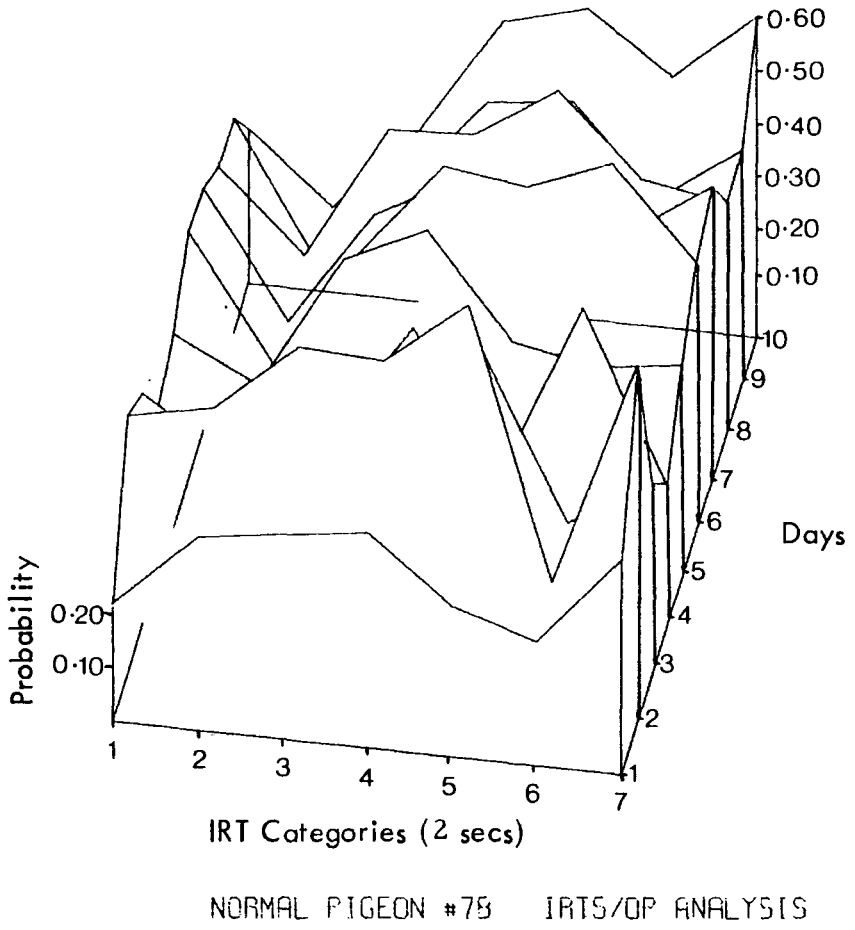


Figure 60. Interresponse times per opportunity analyses on each of the 10 days of DRL 10 training.



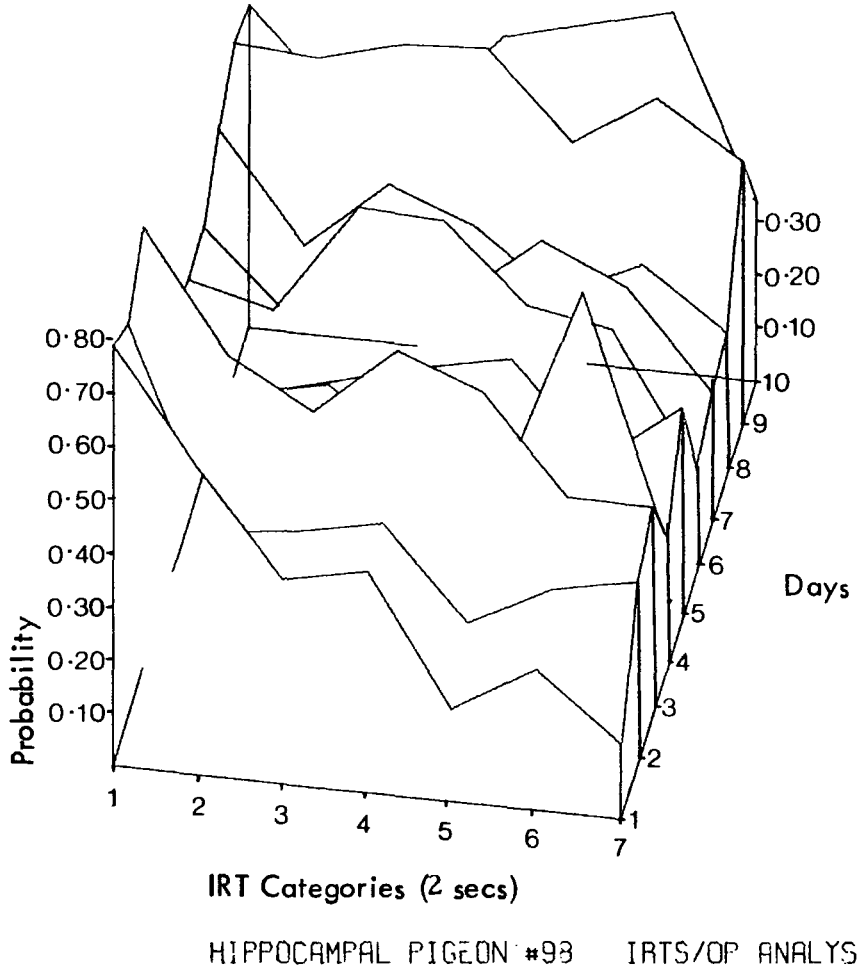


Figure 61. Interresponse times per opportunity analyses on each of the 10 days of DRL 10 training for a single pigeon from each group.

These are presented in Figure 61, and by comparing this with Figure 60 it is clear that certain distortions have occurred in the averaged distribution. Nevertheless, it can also be seen that the main effect has been to smooth the IRT/OP distributions without affecting the overall trends that occur in the two groups over days. Thus, from Figure 61 it can be seen that the normal pigeon developed a more marked bimodal distribution than the hippocampal pigeon, the probability curves for the latter animal generally being flatter than those for the normal pigeon; that the slopes of the probability curves between the first mode and the minimum were generally greater for the normal pigeon; and that the hippocampal pigeon tended to make more responses in most categories, including, to some extent, longer IRTs. Overall, it is clear that the responding of the hippocampal pigeon shown in Figure 61 was under less control of the DRL 10 secs interval than was that of the normal pigeon whose data are presented in this figure. It would seem reasonable to conclude, therefore, that the averaged IRT/OP data are representative of the IRT/OP distributions of the individual pigeons in each of the two groups.

Extinction

1 Responses, IRTs $\geqslant$ 10 secs, and extinction index

During the 20 mins extinction session on the final day of the experiment the measures obtained were the same as those recorded during the extinction trials in the previous DRL 10 task, and as before, a third measure, the extinction index, was derived from these data. These three scores are presented for individual animals in Table 29, and the means are summarised as single data points in Figures 55, 56, and 57. From Figure 55 it can be seen that both groups made virtually the same numbers of responses during extinction as they did during the last day of DRL training. The numbers of IRTs $\geqslant$ 10 secs however, shows a slight decrease for the normal group and a similar increase for the hippocampal group compared with the final day of training. Finally,

Table 29

Extinction session data

Subjects	Normal			Hippocampal		
	Responses	⩾ 10 sec IRTs	Extinction Index	Responses	⩾ 10 sec IRTs	Extinction Index
1	299	20	6.7	462	21	4.6
2	42	27	64.3	418	13	3.1
3	192	38	19.8	858	1	0.1
4	85	26	30.6	465	13	2.8
5	482	14	2.9	494	6	1.2
6	330	11	3.3	523	11	2.1
Means	238.3	22.7	21.3	472.4	12.8	2.3

the extinction index, shown in Figure 57, is seen to be slightly higher than the corresponding efficiency ratios on day 10 in both groups, although the normal group shows the greater increase.

The Mann-Whitney U test was used in the analysis of the comparisons between the two groups on each of these three scores, and it was found that the hippocampal group made significantly more responses and had a significantly lower extinction index than the normal pigeons ($U=3$, $p=0.016$ in both cases), although the smaller number of IRTs$\geq$10 secs achieved by the hippocampal group was only marginally significant ($U=5.5$, $0.064>p>0.042$).

II IRT analysis

a) Relative frequencies of IRTs

The IRTs were again grouped into 1 sec IRT classes and the relative frequency distributions for the two groups are presented in Figure 62. From this it can be seen that neither curve was bimodal. When compared with the relative frequency distributions on day 10 of DRL training (Figure 59) it can be seen that, for both groups, the extinction session did not give rise to an increase in the proportion of burst responses, but the relative frequency of responses in the 1-2 secs IRT class increased noticeably, approximately doubling in each case in extinction. In general, the distributions of both groups shifted marginally towards the shorter IRTs, except that in both cases there was also a slight increase in the proportion of responses in the 20 secs and longer class. In summary, therefore, the hippocampal pigeons again made more short IRT responses and fewer long IRT responses than the normal pigeons.

b) IRTs/OP

As in the IRT/OP analysis of the DRL data, the extinction data were grouped into 2 secs intervals and probabilities were not calculated for those categories in which fewer than 15 responses occurred. The resulting probability distributions for the two

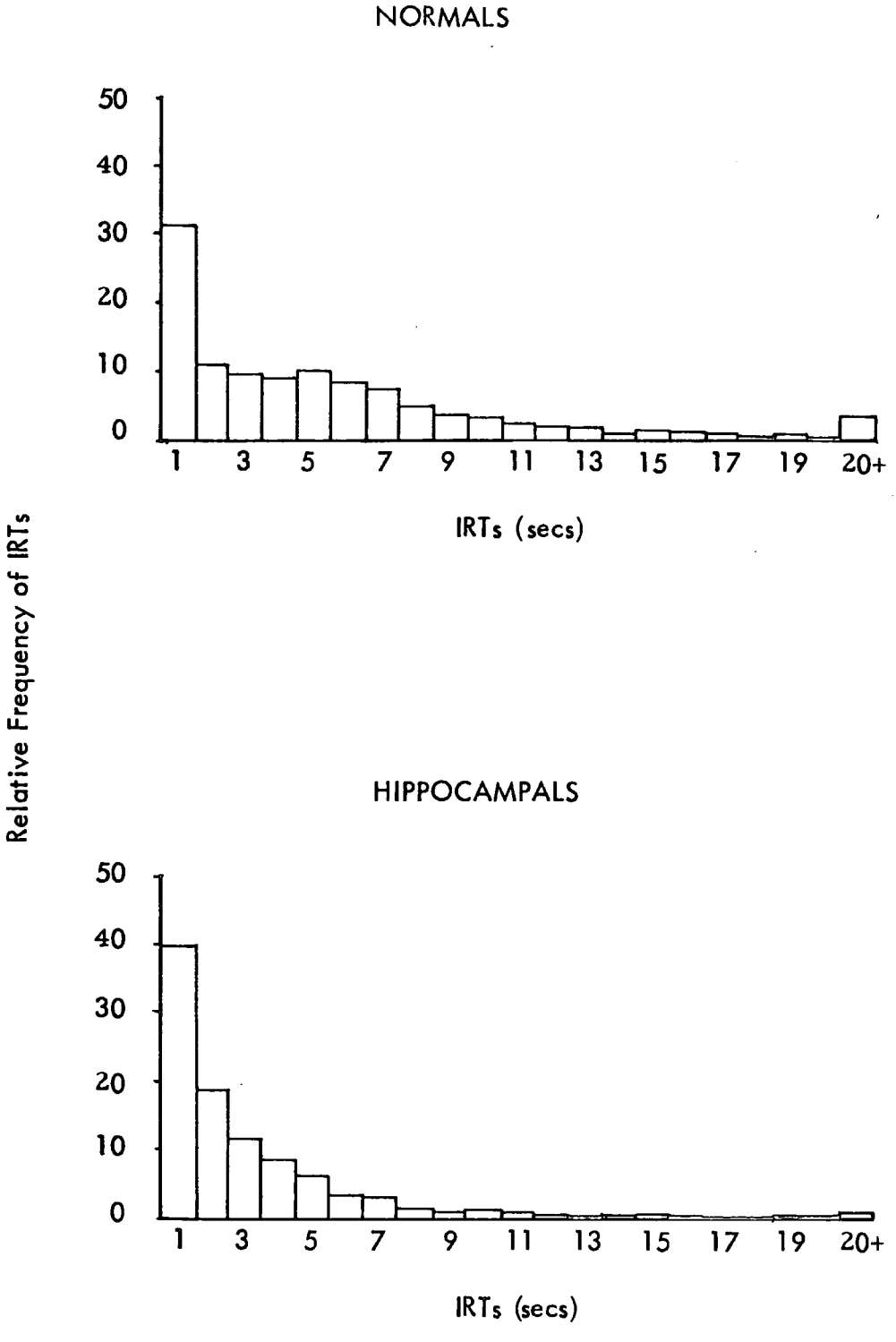


Figure 62. Distributions of the Relative Frequencies of IRTs in Extinction.

groups of pigeons are presented in Figure 63. Here it can now be seen that the response probability distribution for the normal pigeons was bimodal, showing that they maintained a temporal discrimination during the extinction session, although the second mode shifted towards the lower IRT values, the maximum probability of a response now being in the 6-8 secs category. In contrast, the highest response probability for the hippocampal pigeons occurred in the shortest IRT category, i.e., burst responses. Also, the probabilities of a response occurring in any one of the next five categories were approximately equal, indicating that, apart from the burst responses, responding was largely random with respect to time. This therefore shows much more clearly than the relative frequency analysis that the hippocampal pigeons did not maintain any sort of temporal discrimination in extinction. In addition, it can be seen that, compared with the normal pigeons, the hippocampal group showed a greater probability of making a response in any of the 2 secs IRT categories, up to 11.8 secs, and that they made fewer responses with IRTs longer than 11.8 secs, since the numbers of opportunities in each of the longer IRT classes for the hippocampal group were considered to be too small to provide reliable IRT/OP estimates, whereas this was not the case for the normal group.

Discussion

In the previous study of the effects of hippocampal lesions on DRL 10 responding, reported in Chapter 5, it was found that the hippocampal pigeons were impaired on a number of measures. They made considerably more responses and obtained consistently fewer reinforcements throughout training, and consequently they made a significantly lower proportion of reinforced responses than the normal pigeons. During the extinction session that followed DRL training, the hippocampal group made many more responses, achieved fewer IRTs $\geqslant$ 10 secs, and had a much lower extinction index than the normal group. In the present DRL 10 experiment, using a different sample of pigeons, very

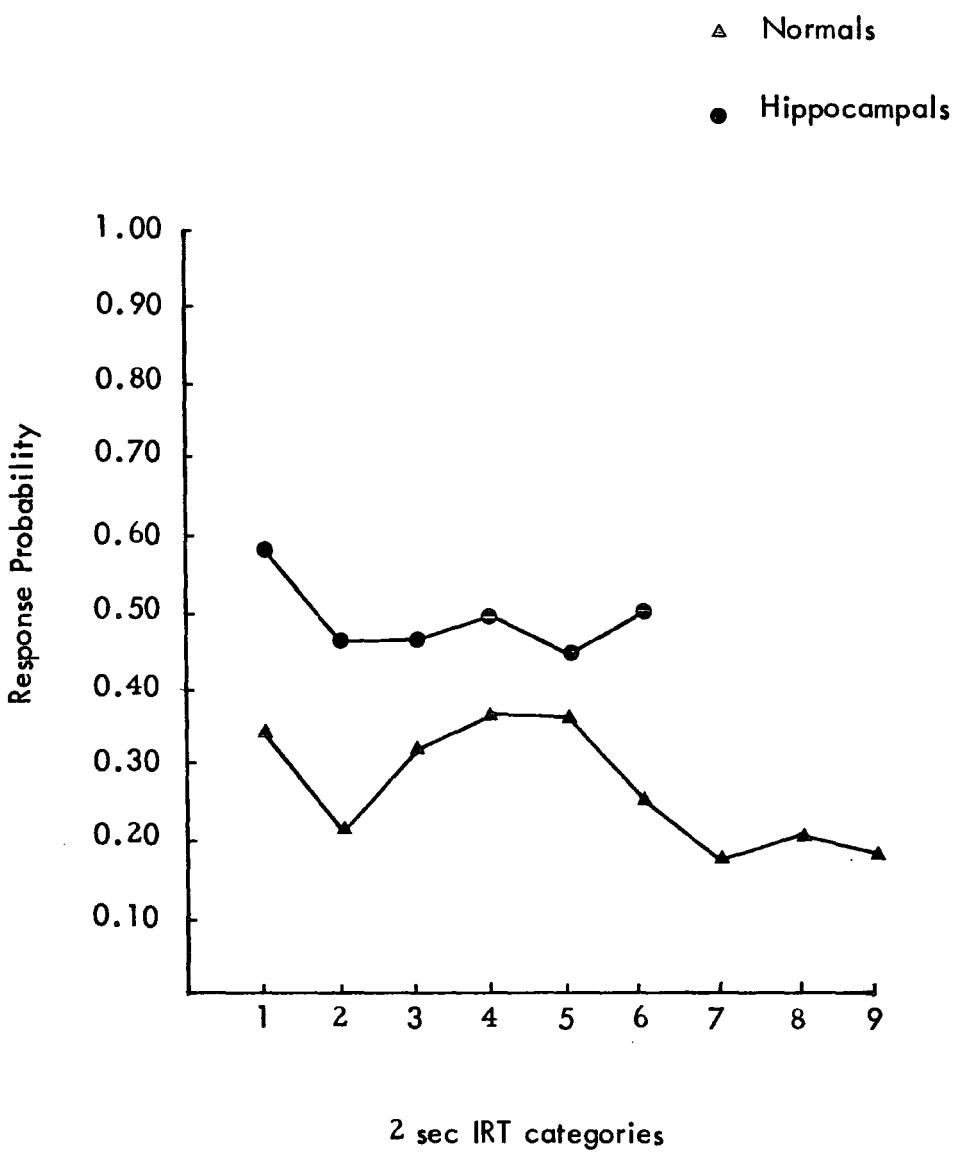


Figure 63. Interresponse times per opportunity analysis of the extinction data.

similar results were obtained, the hippocampal pigeons making significantly more responses and achieving a noticeably smaller proportion of reinforced responses, although, because of daily fluctuations in the scores of both groups, there were no significant differences between the groups in the numbers of reinforcements obtained. Nevertheless, the number of reinforcements obtained each day by the hippocampal group was consistently below that of the normal group. In extinction, the pigeons in the present experiment made more responses, achieved fewer IRTs $\geqslant$ 10 secs, and had a significantly lower extinction index than the normal pigeons. The present results therefore replicate those obtained in the previous DRL 10 experiment, showing that impaired performance on this task by pigeons with hippocampal lesions is a reliable effect, and providing further support for the finding that hippocampal lesions in pigeons produce similar effects on a DRL task to those found in mammals with hippocampal lesions (see Chapter 5).

Kramer and Rilling (1970, pp. 228, 229) have shown that an IRT/OP analysis is a more sensitive measure and can demonstrate the presence of a temporal discrimination which was not shown by a relative frequency analysis. The present data provide a further example which illustrates this by showing that, according to the relative frequency distributions, the hippocampal pigeons were unable to develop a temporal discrimination within ten days of training on a DRL 10 schedule, whereas the subsequent IRT/OP distributions showed quite clearly that a temporal discrimination began to appear after the second day, and that, apart from burst responses, they began to make a major proportion of responses in the 10.0-11.8 secs category. Similarly, the relative frequency analysis of the normal pigeons' responses, although it did detect a temporal discrimination by the fourth day, indicated that the majority of responses, after the burst responses, occurred in the 3.0-3.8 secs category, whereas the IRT/OP analysis showed that a temporal discrimination had begun to appear by the second day, and that

by the seventh day the largest proportion of responses occurred in the 10.0-11.8 secs category, exceeding the numbers of burst responses. Thus, a relative frequency analysis alone would have given rise to the unwarranted conclusion that the hippocampal pigeons had been unable to develop any sort of temporal discrimination within the training period that was available to them, and that the normal pigeons, although showing some sort of temporal discrimination, had been unable to make the majority of their responses in the 10.0+ secs category.

The present results therefore show that both groups of pigeons were able to begin to develop an appropriate temporal discrimination when trained on a DRL 10 schedule even for as short a period as ten days. However, the IRT/OP distributions showed that, compared with the normal pigeons, the hippocampal pigeons were impaired to some extent in their temporal discrimination, in that on most days the proportion of burst responses that they made was greater than the proportion of responses that occurred in the optimum (10.0-11.8 secs) IRT category and greater than the proportion of burst responses made by the normal pigeons. There also appeared to be less discrimination made between the burst responses and responses in the next category (2.0-3.8 secs), and that, generally, the bimodal nature of the daily distributions was less pronounced, indicating that the responses of the hippocampal group tended to be more random with respect to time than were those of the normal group. This result is therefore similar in a number of respects to the impaired temporal discrimination in a DRL task that was reported by Ellen et al (1973) and by Johnson et al (1977), both of whom found that larger numbers of burst responses occurred in rats with combined anterior and posterior hippocampal lesions (Ellen et al) or anterodorsal hippocampal lesions (Johnson et al) than in normal rats. The data of Ellen et al also show that there was a greater tendency for the hippocampal rats to respond randomly with respect to time, even after fifteen days of DRL 20 training. Furthermore, the greater proportion of burst responses made

by the hippocampal animals is consonant with the finding by Ellen and Powell (1962) and Beatty and Schwartzbaum (1968) that rats with hippocampal lesions trained on a F160 schedule were impaired in their ability to develop a typical post-reinforcement pause, and made fewer responses immediately before a reward was due. In Kelsey and Grossman's (1971) terms, the results of the four experiments cited here, and the present results, show that the hippocampal animals made more perseverative errors and fewer anticipatory errors (see Chapter 5, p.177), whereas Kelsey and Grossman found that rats with septal lesions trained on a modified DRL 30 task showed the opposite effect. However, this suggests functional differences between the septal area and the hippocampus, which have also been suggested by the results of other experiments (e.g., Ellen and Powell, 1962; Ellen et al, 1964; Johnson et al, 1977).

It would appear, then, that the analysis of IRTs has provided further clues to the deficit that generally has been found to occur in animals with hippocampal lesions when they are trained on a DRL task following pretraining on a CRF schedule or its equivalent. From the present results it appears that the hippocampal pigeons are capable of developing a temporal discrimination, although it occurs more slowly in these animals than it does in normal pigeons. However, the generally greater proportions of burst responses made by the hippocampal pigeons indicates a greater perseverative tendency immediately following a reward than is shown by the normal pigeons. In their spatial information processing model of hippocampal function, O'Keefe and Nadel (1978) explain the hippocampal impairment on a DRL task by suggesting that normal animals solve the problem by using a place or an orientation hypothesis when responding, and a different hypothesis to avoid the lever or key, this taking the form of what has been referred to as collateral or mediating behaviour. Thus Kramer and Rilling (1970) described a variety of behaviours that have been observed to occur in animals during the DRL interval. One response that was reported to have been observed in pigeons

was that of moving away from the response key to the far corner of the operant chamber, and a similar 'spatial' response has been seen in cats in a similar situation (see O'Keefe and Nadel, 1978, p. 324). Since hippocampal animals are unable to use place hypotheses, they would have to use guidance or orientation hypotheses, but these are said to be inflexible and to lead to persistent patterns of responding. Thus, O'Keefe and Nadel propose that the period of CRF training that invariably precedes DRL training will make it more difficult for the hippocampal animal to adopt a different hypothesis to avoid the lever or key following a reinforcement, whereas the normal animal is able to use a place hypothesis. Because of this, they argue, hippocampal animals should be assisted by the introduction of a cue, and indeed Pellegrino and Clapp (1971) and Rickert, Bennett, Anderson, Corbett, and Smith (1973) both found that the use of a cue caused a reduction in the response rate and an increase in the reinforcement rate of hippocampal rats, enabling them to perform as efficiently as normal rats on the DRL schedule.

Since the present results confirm those obtained in the previous DRL 10 experiment, and showed increased perseverative responding in the hippocampal pigeons following a reward, they confirm that similar deficits on a DRL task occur in hippocampal pigeons as occur in hippocampal mammals.

CHAPTER 10 General Discussion and Conclusions

The experiments reported in this thesis were designed to investigate the behavioural effects of hippocampal lesions in pigeons in order to be able to compare hippocampal function in birds and mammals as inferred from various lesion studies. It was hoped that this comparison would provide behavioural evidence of functional similarities that would support the various findings of structural similarities between the hippocampal formation in the two orders, and therefore also support the proposal, so far made on structural grounds alone, that the avian and the mammalian hippocampus are homologous structures. Moreover, it was hoped that, in the event that similar behavioural effects were found, an analysis of the behavioural changes following hippocampal lesions in pigeons would provide further information, from a comparative point of view, concerning hippocampal function in animals in general.

In the experiments that were carried out in this study, an emphasis was placed on the importance of detailed analyses of the response data that were obtained, since the writer strongly believes that the all-too-common approach of comparing learning or retention in different groups of animals, whether they are intact or brain-lesioned subjects, simply in terms of trials or errors to some particular criterion is often inadequate because it takes no account of possible qualitative differences between groups of animals that have undergone different treatments. This point has also been made recently by O'Keefe and Nadel (1978) with reference to their development of a probe technique which has enabled them to establish that, despite similar rates of learning, normal and brain-damaged animals can show qualitatively different modes of learning.

In the first experiment, which was presented in Chapter 3, it was found that hippocampal pigeons performed more efficiently than normal pigeons on the reversal of a 70:30 colour probability discrimination, and there was the suggestion that they also tended to show greater efficiency on the acquisition of the task. Had the pigeons been

given further training, a clearer effect of the hippocampal lesions on the acquisition of the probability discrimination may have been obtained, and in this respect it is unfortunate that the writer had decided to train the pigeons for a fixed number of days, rather than to train them to criterion. However, as was stated in the Introduction to that experiment, on the basis of experiments reported by others it had been assumed that 2000 trials was sufficient to allow asymptotic, or near-asymptotic levels of performance to be achieved by the pigeons. This experiment therefore needs to be repeated, but with training continuing, in both acquisition and reversal, until asymptotic levels of performance are attained. The greater tendency of the hippocampal pigeons to maximise, at least in reversal, was found to be due to an impaired ability to respond to spatial cues, and in fact these results confirmed a prediction that had been derived from the spatial information processing theory of hippocampal function that has been proposed by O'Keefe and Nadel (1978). Furthermore, evidence was obtained from this experiment which showed that the hippocampal pigeons were not suffering from impaired selective attention, or from a reduced ability to inhibit responding or to shift responses. The second experiment confirmed these findings, since it was found that hippocampal pigeons were impaired on a serial position reversal task because they had difficulty in responding consistently to the appropriate position, but instead shifted their responses between the two keys much more frequently than normal pigeons. However, as in the previous experiment, they did not show an increased resistance to extinction, thereby confirming that the spatial reversal deficit was not due to increased response perseveration to the previously correct position, or to an impaired ability to withhold responses that were no longer rewarded. Furthermore, the nature of their deficit demonstrated clearly that the hippocampal pigeons were not impaired in their ability to shift responses. Nevertheless, in both experiments the hippocampal pigeons showed an increased tendency to persist with particular patterns of responding,

and this was investigated further in the third experiment, reported in Chapter 5 and with a replication presented in Chapter 9, in which normal and hippocampal pigeons that had been trained on other, dissimilar, tasks with CRF or low FR schedules of reinforcement were then trained on a DRL 10 schedule of reinforcement. In both experiments clear evidence was obtained which showed that the hippocampal pigeons were much more persistent than the normal pigeons in their use of previously acquired response patterns which were incompatible with efficient performance on a DRL schedule, since they consistently made many more responses and received fewer reinforcements. In the second DRL 10 experiment IRTs were also recorded, and the detailed analysis of them which was undertaken suggested that the timing behaviour of the hippocampal pigeons was different from that of the normal pigeons, in that the hippocampal animals were less able, or were slower than the normal animals, to develop a temporal discrimination. In the fourth experiment, which was reported in Chapter 6, it was found that the hippocampal pigeons were able to learn both the acquisition and the reversal of a simultaneous visual discrimination at normal rates, but detailed analysis of the individual responses indicated that the hippocampal pigeons were using different strategies from those used by the normal pigeons, and also that they tended to make fewer position responses and to use fewer position hypotheses. A prediction concerning the effects of overtraining on reversal learning in hippocampal pigeons, that was derived jointly from Mackintosh's analysis of the ORE in rats and birds (e.g., Sutherland and Mackintosh, 1971) and the spatial information processing model of hippocampal function, was not confirmed, but it seems most likely that this was due to the nature of the visual task that was used in this experiment in that it was not as simple a task as preliminary tests had indicated. Clearly, this experiment should be repeated, but with a brightness or a colour discrimination in place of the simple form discrimination that was used here. The

final two experiments, presented in Chapters 7 and 8, showed that hippocampal pigeons are not impaired on the acquisition of either a spatial or a nonspatial alternation task in an operant chamber, even with delays of up to 10 secs between trials. Reviews of many of the studies of the behavioural effects of hippocampal lesions in mammals (mainly rats, although there have been a number of experiments reported in which monkeys were the subjects, and also some involving cats) have been presented throughout this thesis, and for each of the experiments reported here comparable results have been obtained in mammals with hippocampal lesions.

In the Introduction to the studies presented here reference was made to the variety of functions that have been assigned to the hippocampus in order to explain the various behavioural changes that have been found to result from hippocampal lesions in mammals. However, it was also noted that, in a sizeable number of studies results were obtained that were inconsistent with any of the inhibition hypotheses of hippocampal function, regardless of whether they were concerned primarily with response-inhibition (Kimble and Kimble, 1965; McCleary, 1966; Altman et al, 1973) or with the inhibition of attention (Douglas and Pribram, 1966; Douglas, 1967; Kimble, 1968; Silveira and Kimble, 1968; Pribram et al, 1969; Kimble and Kimble, 1970). Both Douglas et al and Kimble et al had also suggested that the hippocampus is involved in the regulation of hypotheses, although in both cases this mechanism was seen to be closely related to the proposed selective attention function of the hippocampus. Furthermore, the finding that hippocampal mammals are capable of normal acquisition and reversal performance on nonspatial discrimination tasks (e.g., Isaacson et al, 1968; Mahut, 1971; Jones and Mishkin, 1972; Samuels, 1972), and are not impaired on the acquisition of a spatial alternation task in either an operant chamber (Stevens and Cowey, 1972, 1973) or a WGTA (Brown et al, 1969; Waxler and Rosvold, 1970) is incompatible with the suggestion that the hippocampus is involved in a response-shift mechanism (Olton, 1972a).

In an attempt to resolve the apparent discrepancy between the human and the animal data, Weiskrantz (1971) and Weiskrantz and Warrington (1975) argued that hippocampal lesion deficits in all mammals could be explained in terms of interference effects which were seen to impair the retrieval of appropriate information or the selection of appropriate responses, since it had been found that the use of partial cueing techniques assisted the retrieval of correct responses by amnesic patients. Moreover, they proposed that much of the animal data could also be explained in similar terms, and subsequently they showed that the use, in the amnesic patients of certain experimental paradigms, that were similar to those that had been used with hippocampal animals gave rise to similar deficits in the human subjects (see Weiskrantz and Warrington, 1975). Recently, two experiments by Winocur (Winocur and Black, 1978; Winocur, 1979) provided evidence for the interference hypothesis of hippocampal function in rats trained in a passive avoidance task and a visual discrimination experiment. However, there is sufficient evidence from other reports of experiments with rats on, for example, reversal learning, successive go, no-go discriminations, and extinction, which shows that hippocampal animals are not necessarily impaired on tasks which require them to make responses that are incompatible with previously acquired responses. Furthermore, Winocur (1979) had reported that the hippocampal and the normal rats in the visual discrimination task had apparently used similar response strategies in order to learn the discrimination, although there are numerous reports which show that different response strategies, or hypotheses are used by hippocampal animals (e.g., Silveira and Kimble, 1968; Kimble and Kimble, 1970; Isaacson and Kimble, 1972; O'Keefe and Nadel, 1978), particularly in tasks involving spatial cues. Similar findings were also obtained in the present experiments, although the hippocampal pigeons were not impaired in extinction or on the reversal of a nonspatial discrimination. It was noted above (see also the Introduction, pp. 5-6)

that, in proposing the interference hypothesis of hippocampal function, an attempt was made to reconcile the apparently discrepant effects of hippocampal damage in man and in lower animals. However, recently it has been extensively argued by Horel (1978) that hippocampal lesion effects in animals and the amnesic syndrome in man are not comparable because they involve damage to different parts of the brain. Thus, Horel has proposed that the region in the human brain that, when damaged, is much more likely to be responsible for the amnesic effects is the so-called temporal stem, or albal stalk, and adjacent parts of the temporal cortex that are invariably included in damage involving the hippocampus, and in confirmation of this proposal Horel reports that monkeys with lesions of the temporal stem and cortex, but with the hippocampus left intact, showed severe learning and retention deficits similar to those that occur in amnesic patients.

Throughout the studies reported here, reference has been made to an alternative explanation of the effects of hippocampal lesions in animals, the spatial information processing, or cognitive mapping model of hippocampal function that recently has been formally proposed by O'Keefe and Nadel (1978), although less complete versions had been published earlier (e.g., Nadel and O'Keefe, 1974). The importance of spatial cues in the hippocampal deficit had previously been noted by Mahut (1971) and Samuels (1972) and particularly marked deficits in the use of spatial information by hippocampal rats and monkeys in various tasks have been reported by Olton and Isaacson (1968), Cohen et al (1971), Mahut and Zola (1973), Plunkett et al (1973), O'Keefe et al (1975), Olton et al (1978), and Sinnamon et al (1978), and were also found in the present experiments with hippocampal pigeons. It is proposed, therefore, that the results of the present experiments provide good evidence for the existence of functional similarities between the mammalian and the avian hippocampus, and thus for the proposal that they are homologous structures. Moreover, these results

appear to be most consistent with the hypothesis that the hippocampus is involved in the processing of spatial information as proposed by O'Keefe and Nadel (1978), and it is of interest that O'Keefe and Nadel suggest (p. 104) that "it is reasonable to assume that behaviours such as homing, migration, and territoriality are evidence of cognitive mapping and suggest as a working hypothesis that species demonstrating these behaviours have a homologue to the mammalian hippocampus". Since these behaviours are typical of birds, the present findings provide support for this working hypothesis. Arising from this, of course, is the prediction that homing pigeons given hippocampal lesions should be greatly impaired in their subsequent ability to home. The present study has therefore provided evidence of further similarities between the avian and the mammalian brain that extend the findings of Karten and Hodos and their colleagues, at the same time confirming the suggestion of Macphail (1975b) that the study of a species whose brain organisation differs from that of mammals may provide useful clues to the understanding of the mammalian brain.

As a final point, it must be recognised that the use of lesion techniques for the study of normal brain function has its critics (e.g., see Weiskrantz, 1973), although in the view of the writer it would appear to provide a very useful starting point from which to work; but it is also maintained that subsequent investigations should approach the problem of understanding brain function from as many different angles as possible. In a sense, such a viewpoint has already been advocated, in that it has been argued that the establishment of homology in the nervous system requires evidence from both structural and functional studies. It is therefore proposed that, in addition to the further lesion studies that need to be carried out in order to extend the present findings, evidence is also required from electrophysiological studies, particularly of single cell responses to spatial cues similar to those that have been carried out by O'Keefe and Dostrovsky (1971), Ranck (1973, 1975), and O'Keefe (1976).

APPENDIX 1 Summary of data and calculations for the box-and-whisker plots.

Summary of data for box-and-whisker plots with 95% confidence intervals for the position responses of normal and hippocampal pigeons during acquisition (see Chapter 3).

Hippocampal group

Summary of data:

N = 80

M	40	60.5	
H	20	53	74
		35	95

Depth of median = 40

Depth of hinges = 20

Median = 60.5

Hinges: upper = 74

lower = 53

Extremes: upper = 95

lower = 35

Normal group

Summary of data:

N = 80

M	40	59	
H	20	51	65
		34	78

Depth of median = 40

Depth of hinges = 20

Median = 59

Hinges: upper = 65

lower = 51

Extremes: upper = 78

lower = 34

Standard deviation of the median, s,

$$= \frac{1.25R}{(1.35\ N)}$$

where R = interquartile range

N = number of scores

R = 74 - 53 = 21

N = 80

$\therefore$ s = 2.17 (68% confidence level).

95% confidence level = $1.96s$ = 4.26

$\therefore$ M + $1.96s$ = 64.76

M - $1.96s$ = 56.24

R = 65 - 54 = 11

N = 80

$\therefore$ s = 1.45 (68% confidence level)

95% confidence level = $1.96s$ = 2.84

$\therefore$ M + $1.96s$ = 61.84

M - $1.96s$ = 56.16

APPENDIX 2 Stimulus presentation sequences.

APPENDIX 2A

Stimulus Presentation Sequences Used in the Colour Probability Experiment

$\underset{o}{L}$ = Majority colour on the left key and reinforced

$\underset{o}{R}$ = Majority colour on the right key and reinforced

$\underset{*}{L}$ = Majority colour on the left key, but minority colour reinforced

$\underset{*}{R}$ = Majority colour on the right key, but minority colour reinforced

SEQUENCE 1	SEQUENCE 2
1. L L R L R R L L R R o o o o * o * o o *	11. R L R R L L L R R L * o o o o * o * o o
2. R L L L R R L R R L o o * o o o o * o *	12. L R R L L R R L L R o * o * o o o o * o
3. L L R L R R R L L R o o o o * * o * o o	13. R R L R L L L R R L * o * o o o * o o o
4. R L R R L L L R R L * o o o * o o o * o	14. L R R L R R L L L R o * o o o o * o o *
5. L L R R L L R L R R o * o o o o * * o o	15. R R L R L L R R L L o o o * * o o o o *
6. R L L R L L R R R L * o o o o * o * o o	16. L R R R L L R L L R o * o * o o o * o o
7. L L R R L L R R L R o * o o o o * o * o	17. R L L R R R L L R L o * o o o * o o o *
8. R L R R L L R R L L * o o o * o o o * o	18. L R L L R R R L L R o o o o * o o o * *
9. L R L L R R L L R R o o * o * o o o * o	19. R L L R R R L R L L o * o o * * o o o o
10. R L L R R L L R R L o o * * o * o o o o	20. L L R R L R L L R R * o * o o o o * o o

APPENDIX 2A (continued)

SEQUENCE 3

21. R R L R L L R R L L
    o o o * o * o o o *

22. L R R R L L R L L R
    o * o o o o * * o o

23. R R L R L L L R R L
    o o * * o o * o o o

24. L R L L R R R L L R
    * * o o o o * o o o

25. R R L L R R L R L L
    * o o * o o o o * o

26. L R R L R R L L L R
    o * o o o * o * o o

27. R R L L R R L L R L
    o * o o o o * o o *

28. L R L L R R L L R R
    * o o o * o o o o *

29. R L R R L L R R L L
    o o * o o * o o o *

30. L R R L L R R L L R
    o * o o o o * o * o

SEQUENCE 4

31. R R L L R L R R L L
    o * o * o o o o * o

32. L R R L R L L L R R
    o o * o o * o o o *

33. R L R R L L L R R L
    * o o o * o o o * o

34. L L R R L R L L R R
    o * o o o o * o o *

35. R L R L L R R R L L
    * o o * o o * o o o

36. L R R L L R L L R R
    * o o o * o o o o *

37. R L L R R R L R L L
    o * o o * o o o o *

38. L R R L L L R R L R
    o * o o o * o o o *

39. R R L L R L L R R L
    o o o * * o o o o *

40. L L R R R L R L L R
    o o o * o * * o o o

APPENDIX 2B

Autoshaping Schedule

The centre key is illuminated on the first trial of each day, and thereafter on every third trial, the intervening two trials being distributed on the two side keys according to a Gellerman sequence as follows:

(L = left key, C = centre key, R = right key)

1	C R R C L R C L L C R R C L L	C L R C R R C L L C R L C L R
2	C R R C L R C L L C L R C R L	C L R C L L C R R C R L C L R
3	C R R C L L C R R C L R C L L	C L R C R L C R R C L L C L R
4	C R R C L L C R R C L L C R L	C L R C L L C R R C L L C R R
5	C R L C R R C L L C R R C L L	C L R C R L C L R C R L C L R

Stimulus Presentation Sequences Used in the Visual Form Discrimination Task

(L = left key, R = right key, and specify the key on which the correct stimulus was presented on each trial.)

1. R R L R L L R R L L
2. L R R R L L R L L R
3. R R L R L L L R R L
4. L R L L R R R L L R
5. R R L L R R L R L L
6. L R R L R R L L L R
7. R R L L R R L L R L
8. L R L L R R L L R R
9. R L R R L L R R L L
10. L R R L L R R L L R
11. R R L L R L R R L L
12. L R R L R L L L R R
13. R L R R L L L R R L
14. L L R R L R L L R R
15. R L R L L R R R L L
16. L R R L L R L L R R
17. R L L R R R L R L L
18. L R R L L L R R L R
19. R R L L R L L R R L
20. L L R R R L R L L R
21. R R L L L R R L R L
22. L L R R R L L R L R
23. R R L L L R L R R L
24. L L R R L R R L L R
25. R L L R R R L L R L
26. L L R R L L R R L R
27. R L L R R L R R L L
28. L R R L L L R L R R
29. R L L R R L L R R L
30. L L R R L L R L R R
31. R L L R L R R R L L
32. L R L R R L L L R R
33. R L L R L L R R R L
34. L L R L R R R L L R
35. R L L L R R L R R L
36. L L R L R R L L R R

APPENDIX 2D

Stimulus Presentation Sequences Used in the Colour Alternation Experiment

A = Green on left and reinforced

B = Red on right and reinforced

C = Green on right and reinforced

D = Red on left and reinforced

SEQUENCE 1

1. C D C B A B C D A B
2. A D C D A B C B A D
3. C B C D A B A D C B
4. A D A B C D C B A D
5. C D A D C B A D A B

SEQUENCE 2

1. A D C B C D A B A D
2. C D A B C D A B C B
3. A D A B C D A B C D
4. C B C D A B C D A B
5. A D C B A D C B A D

SEQUENCE 3

1. D C B A B C D C B A
2. B C D A B C B A D C
3. D A D C B A B C D A
4. B C B A D C B A D C
5. D A B C D A D C B A

SEQUENCE 4

1. A D C B A D A B C D
2. C B A D C D A B C B
3. A D C B A B C D A D
4. C D A B C B A D C B
5. A B C D C B A B C D

SEQUENCE 5

1. B A D C B C D A B C
2. D A B C D C B A D A
3. B A D C B A D C B C
4. D A B C D A D C B A
5. B C D A B A D C B C

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